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ABSTRACT

The purpose of the course on aphasia in adults, from which these proceedings resulted, was to increase the knowledge and skill of professional persons who are actively engaged in the areas of aphasia: in research, rehabilitation, or teaching. The course was jointly sponsored by the University of Virginia and the Vocational Rehabilitation Administration, and was presented at the University of Virginia and the Woodrow Wilson Rehabilitation Center on December 3-6, 1963. The first section of this compilation, "The Nature of Aphasia," contains papers by J. Eisenson, W.G. Hardy, H. Goodglass, W.E. Castle, and W. Reise. The second section, "The Evaluation of the Aphasic Adult," contains papers by F. Dreifuss, J. Eisenson, J. Lore, and H. Goodglass. "The Treatment of the Aphasic Adult," the third section, contains papers by C. Reedy, J.H. Allan, R.M. Hoover, H. Goodglass, J. Eisenson, P. Breeding, M. Taylor, and R. Stoudt, and a joint evaluation of progress by J. Eisenson, W.G. Hardy, and M. Taylor. "Directions in Research on Aphasia," the final section, contains commentaries by F. Dreifuss, H. Goodglass, M. Taylor, W.G. Hardy, and J. Eisenson. A roster of participants concludes the proceedings. [Not available in hard copy due to marginal legibility of original document.] (AMM)

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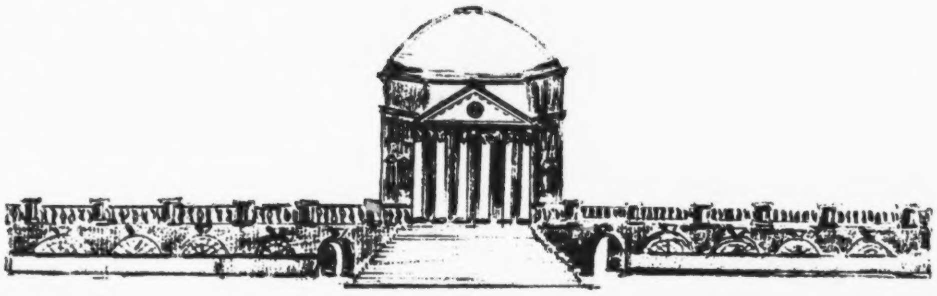

THE APHASIC ADULT

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Evaluation
and
Rehabilitation

Edited by Helen G. Burr

Proceedings of a Short Course
December 3 - 6, 1963
University of Virginia
Charlottesville, Virginia

Jointly Sponsored By
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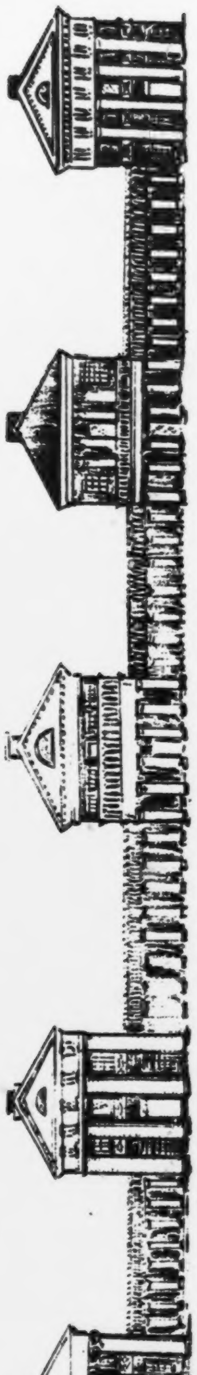

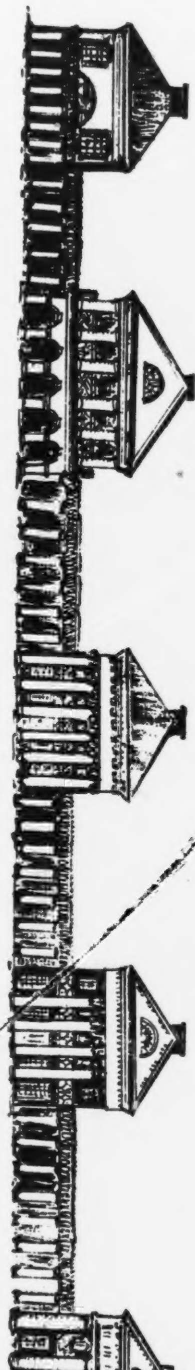

The "Academic Village" of Mr. Jefferson's University

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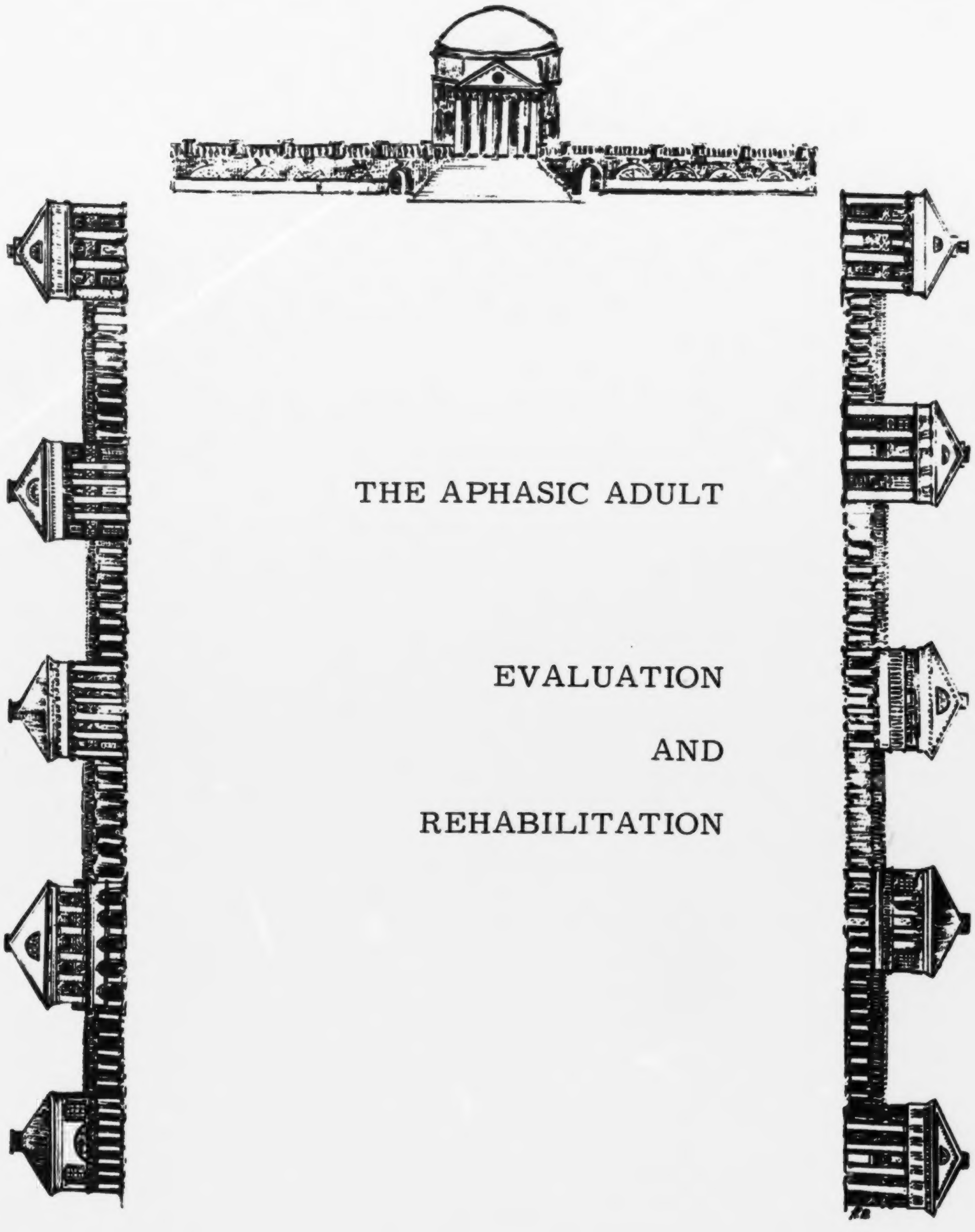

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PREFACE

The purpose of the course on aphasia in adults, from which these proceedings result, was to increase the knowledge and skill of professional persons who are actively engaged in the area of aphasia: in research, rehabilitation, or teaching. The course was jointly sponsored by the University of Virginia and the Vocational Rehabilitation Administration, and was presented at the University of Virginia and the Woodrow Wilson Rehabilitation Center on December 3 through 6, 1963.

The unedited proceedings--consisting of 426 pages--were reduced by more than one half, to conform with provisions of the publication grant. The abridgement process involved elimination of such portions of the record as introductions of speakers and topics, demonstrations of visual materials, and discussions following prepared speeches. Pertinent points from informal discussion were incorporated into the formal presentation, wherever possible, to avoid loss of substantive content. Loss of the form and flavor of extemporaneous comment was unavoidable.

A great many people cooperated in making the course and this publication possible. Particular mention must be made of the following: Mrs. Betty Lawrence, who transcribed the tape recordings; Dr. Ralph Bralley, who served as Coordinator of the course; Mr. James Bryden, Jr., who read proof; Mr. and Mrs. Julio Suarez-Galban, Mr. and Mrs. Lewis Marran, and Mr. and Mrs. James McCahill, who opened their hearts and homes to the participants for special events; the Vocational Rehabilitation Administration, who supported the course and publication of the proceedings; the visitors and members of the short course faculty, whose knowledge and whose thoughtful, thorough preparation made the course a success.

H. G. B.

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OPENING REMARKS AND WELCOMING ADDRESS

OPENING REMARKS

Helen G. Burr

One of the most serious problems confronting the field of speech and hearing today is the need for provision of concentrated postgraduate educational opportunities to practicing speech pathologists and audiologists. In our field, as in other scientific fields, there is an increasing gap between available knowledge and the application of that knowledge in clinical practice and teaching.

A great deal has been said, and is being done, about relieving the acute shortage of personnel in speech and hearing. Until recently, however, little national emphasis was given to improving the quality of present programs. With the realization that continuing education is synonymous with good teaching and good clinical practice, several governmental agencies are sponsoring a number of short, postgraduate courses for speech pathologists and audiologists. The University of Virginia is very pleased to co-sponsor this course on aphasia with the Vocational Rehabilitation Administration of the Department of Health, Education, and Welfare.

Planning began in March, 1963, when the Vocational Rehabilitation Administration invited this University to conduct a postgraduate course in some professional aspect of our field. In that month, we sent out a questionnaire for the purpose of determining the area of greatest professional concern to 175 of our colleagues in Virginia. The majority of respondents expressed a primary interest in aphasia. It soon became evident that this concern in Virginia, with the adequacy of our present programs for aphasics, also prevailed throughout the nation. Announcement of the course on aphasia brought requests for further information from individuals in 35 states, Canada, Puerto Rico, and the District of Columbia--and for every five applications for Traineeships that were received, only one could be awarded. We are, therefore, particularly pleased that the Vocational Rehabilitation Administration is supporting publication of the proceedings, so that the substance of our deliberations together, during these four days, can be shared with all who wished to be here.

I should like to invite all of you to visit the Speech and Hearing Center while you are at the University of Virginia, and I urge you to walk through the grounds of the University--particularly the central and historic portion known as the Lawn. We hope your memories of the University and your associations here will be as valuable to you personally as the ideas here generated will be to aphasia rehabilitation in your respective centers and institutions.

Charlottesville, besides being the home of the University of Virginia, is the headquarters of Region III of the Department of Health, Education, and Welfare. Region III includes Virginia, West Virginia, Maryland, North Carolina, Kentucky, Puerto Rico, the Virgin Islands, and the District of Columbia. We are most fortunate in having with us this morning--to welcome us and to begin our program--the Director of Region III, Mr. Edmund Baxter.

WELCOMING ADDRESS

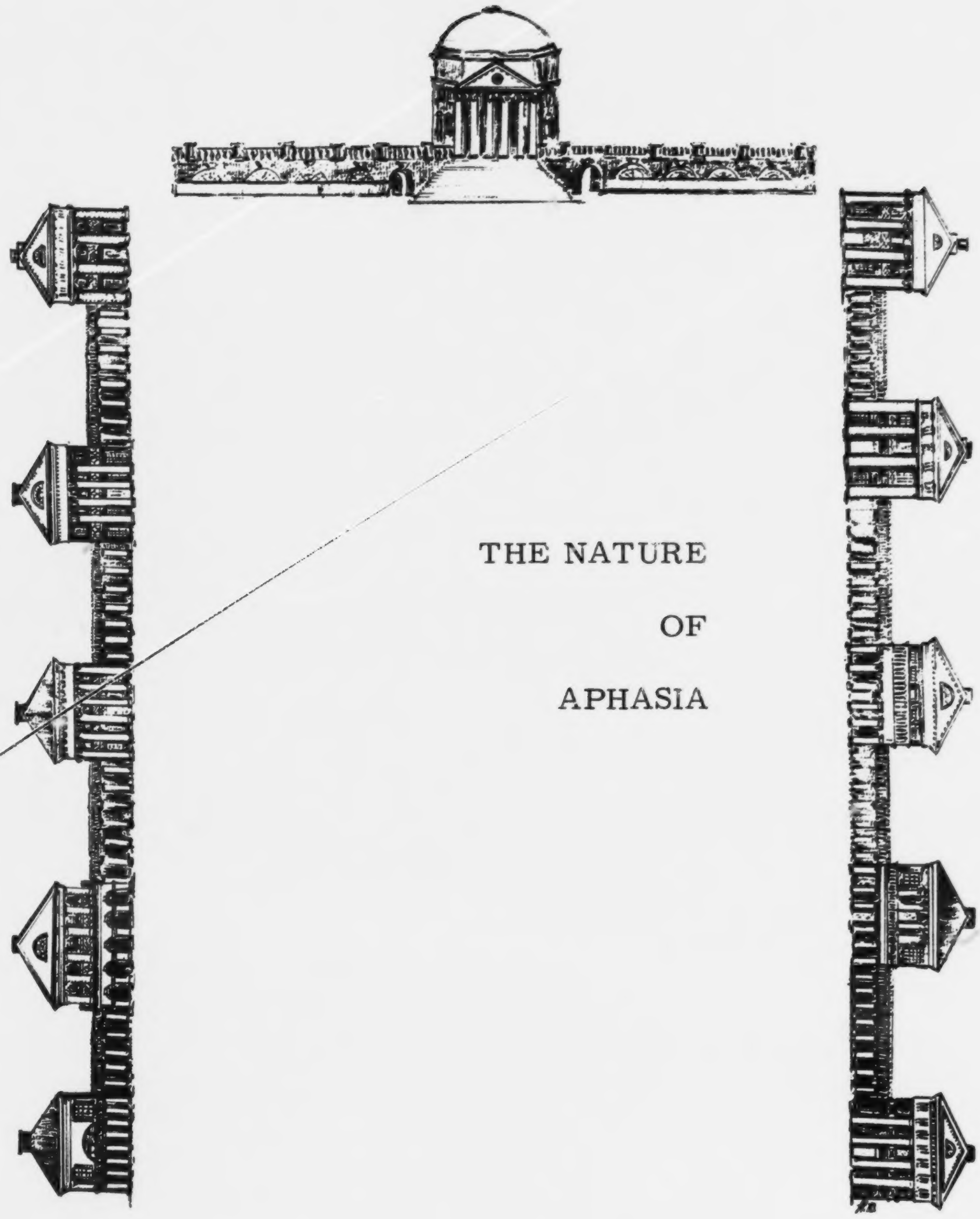
Edmund Baxter

I think it is obvious already this morning that Dr. Burr, my friend Helen, is an authority on speech. Anyone who can describe the territory of Region III, with all of those tongue twisters, is quite good. I am reminded, also, of how awkward and clumsy our name is--the Department of Health, Education, and Welfare. In fact, on one occasion when I was making a visit to a community which is thought of often as part of the Bible belt, I was introduced as the Regional Director of the Department of Wealth, Education, and Hellfire.

Thank you, Helen, for the opportunity to be here among this distinguished and dedicated group of people. It is more than a privilege and a pleasure to share with you and my friend Mr. Reedy and your staff the opportunity to welcome this excellent group. I hope all of you will enjoy your stay in Charlottesville and the University atmosphere while you are pursuing this serious work and this interesting opportunity to enlarge your professional knowledge in behalf of helping a seriously handicapped group of people. I think it is both appropriate and significant that this conference should be held in the delightful and inspiring atmosphere of the University, founded by the ingenious and resourceful Mr. Jefferson. We, in the Department of Health, Education, and Welfare, always welcome the opportunity to join with the University in the many programs in which we share a mutual interest.

I would like to be wise and profound, but in this group I am sure I would be in deep trouble if I made that attempt. I would like to say that, although only a small minority of our total population is included in the group of people who are described as having aphasia problems, this is one of the challenging and critical areas confronting us today. I think we are lagging far behind in our research in services and, perhaps most important, in the marketing of research in this field. It happens that I have considerably more interest in this subject than that of a mere administrator, but in the administrative process I am constantly reminded of the vital necessity of free, full, and open communication. Of course, one of your goals in this conference is to promote effective communication. Some of us who are not victims of physical or emotional problems in relation to communication obviously are not taking full advantage of our faculties and facilities, but this is beside the point, except as it does call to our attention the extreme importance of communication and as it also calls to our attention one of the major purposes of this conference--the need for an interchange of information throughout the disciplines and professions concerned with aphasia.

I think it is clearly the role of government, and especially the Federal government, to lend vigorous and active support to your determined efforts. I hope that your conference will prove to be both enlightening and productive, and I have every confidence that it will. Those of us with the Department of Health, Education, and Welfare, especially the Regional Office, invite you to call upon us at any time for assistance or help that you think we can give--both in relation to the work of this conference and to your future efforts. In closing, I would like to suggest that we try here to take full advantage of our opportunity to enlarge communication with each other and that we do it with the same sense of purpose we all advocate in combating the communication problems which are inherent in aphasia. I thank you for this opportunity and best wishes to all of you for a successful conference.



THE NATURE
OF
APHASIA

CHARACTERISTICS OF APHASIA IN ADULTS

Jon Eisenson

As a human problem amenable to treatment, aphasia is a comparatively new field of study. Because of its tremendous import, because of its interest, because of its romance, because of the personalities involved (patients, clinicians, and students), aphasia is able to compete with the field of stuttering in filling lecture halls. If you attend the convention of the American Speech and Hearing Association, or any regional convention, you may well find that there are almost as many people eagerly waiting to learn something about aphasia as there are people waiting to learn something about stuttering. Stuttering is mentioned in the Bible, is defined in the Bible, even its causes are implied in the Bible--but aphasia is not. And so, stuttering has several thousand years of advantage over the field of aphasia in regard to arousing curiosity in people. In regard to the field of stuttering, as in the field of aphasia, we are perhaps beginning to learn what questions to ask in order to find out what the enigma is about. Had we stopped 40 years ago in our study of aphasia and aphasics, we would have been relatively smug and secure. Forty years ago we knew how to define aphasia and we thought we knew what aphasia was about. If we were to fix the date for terminating our studies of aphasia at the end of World War I, rather than World War II, we would have a set of answers about which we might have felt relatively secure. We had very few patients who could confuse the issues or clinical conclusions by their living presence. Aphasic persons were what a handful of neurologists said they were in textbooks, or in a few articles that were written by neurologists. The articles--however few--outlived the patients. So, if you look through textbooks on neurology in the quarter century before World War II you will note there is very little change in definitions or concepts of aphasia or aphasic patients. Aphasics were what the neurologists said they were and there were no patients to refute their concepts. There were very few patients that younger and less prejudiced clinicians could study at first hand. If you wanted to know what Broca's aphasia was, you read Broca. If you happened to be in France, you might have gone to the jar that contained the brain of one of Broca's patients. Broca, of course, did not know anything about American concepts of statistics and numbers. On the basis of an unbroken series of two patients, Broca came to very firm conclusions about aphasia. Possibly Broca's colleagues urged him on to earlier and firmer conclusions than Broca held himself and Broca, impressed, did not argue with his colleagues. Later on, an upstart did raise the questions about Broca's patients, and one even took the trouble to examine the brain and found, to be sure, that the brain of Broca's first patient did have pathology where Broca said it was to be found--but there was also evidence of lesion in areas that Broca had not observed, or at least not noted.

The situation in regard to aphasia is a bit different now. Before World War II, aphasia was almost exclusively an area of pathology reserved for the neurologists. Since World War II, the field of aphasia has been opened wide to students of other disciplines. In the United States and abroad, psychologists and speech pathologists have become concerned not only with aphasia as an area of study but with aphasics as persons to be studied and treated and rehabilitated, if possible. Today aphasia and aphasics are the subjects of study of representatives from the fields of linguistics, psycholinguistics, statistics, neurophysiology, neuropsychology, philosophy, psychiatry, and, oh yes, even a few persons who are interested in what we can do with all of this information in helping the aphasic to recover from his impairment. What are the impairments? I thought that in the preparation of this talk I might ask a few experts on the subject what they considered their impairments to be, but I did not go to the experts who write books and articles. I went to the experts who had personal experience with the problem--direct experience rather than value judgments. I went to the aphasics themselves; to persons who had made a pretty good recovery, who could do most or the whole of Wepman's examination with relatively few errors, and who could complete my inventory in an hour with no more than two or three errors. I consider these to be fairly well recovered patients. I got a variety of judgments from them as to what aphasia was, to them. They were not concerned with textbook considerations or with diagnostic or prognostic implications. They were not concerned with whether

Joseph Wepman was correct, or Hildred Schuell was correct, or whether Broca had the right idea in the first place, or whether Kurt Goldstein was closer--in his concept of aphasia--to what was disturbing them when they were patients. They were telling me what aphasia meant to them. Here are some of their evaluations. "My aphasia was not being sure of what I had to say. It was not being sure of what I heard so I had to beat around the bush until I figured it out and had to hope that I could find the right words to say when I did figure things out, only I never could be sure, and I'm not sure today."

The second expert told me: "Aphasia is not finding the right words or not knowing whether I could find just the right words and put them together in just the right way." Another opinion: "Aphasia was frustrated thinking and being frustrated in telling others what I thought. I got the ideas okay, and if only I would have done something instead of saying something I would have stayed out of trouble. Aphasia happens when you have to tell people instead of having to do something. Aphasia is not knowing what in the hell I should say when someone asks me a question, even though I always knew, or thought I knew, what I should say if the question was addressed to somebody else." Another point, another patient, another expert: "Aphasia is the difficulty I had in getting along with people, with my wife and with my kids, because they thought I had become feeble-minded. It was trying to keep control of myself when I became angry because people thought me stupid. It was also a terrible fear, a suspicion that maybe they were right." Another point: "Aphasia is finding myself saying, or thinking, or doing something over and over again when I know I shouldn't." Is there a common core to all of these? I think there is. Each of these experts, each person who had been an aphasic and was admittedly still having some of the residual effects, was telling what aphasia was to him. I do not know whether we can take these answers and put them into slots, or put them into categories, or feed them into machines and have the machines tell us what aphasia is. Sometime during the course of this workshop you will learn that this is now an approach to the study of aphasia; this is pretty much how the psycholinguists are trying to study the problem. Aphasia is going to be in part, what the machines tell us aphasia is, but the machines, as yet, do not contain entire aphasics. They just contain responses made by aphasics. I do not know any machine that has yet been devised to take into consideration the particular circumstances that motivate the particular response a patient produces at a given moment, that will later be fed to the machine for digestion, assimilation, and presumably for generalization. All of this to help us to learn what aphasia is.

At this point I think it should be helpful to consider one or two contemporary definitions of aphasia. Penfield and Roberts[1] define aphasia as "that state in which one has difficulty in speech, comprehension of speech naming, reading, and writing, or any one or more of them; and it is associated with misuse and/or perseveration of words, but is not due to disturbance in the mechanism of articulation (as in pseudo-bulbar palsy) or involvement of peripheral nerves, nor due to general mental insufficiency." Impairment in the use of gesture should be added to those in the comprehension of oral and written words.

Another way of viewing aphasia is to consider the disturbances, and the patient, in terms of the kind of verbal behavior one may expect according to a given set of circumstances. So, on a statistical basis, aphasia may be considered as a reduced likelihood that a given linguistic reaction, receptive-evaluative or productive, will take place in kind and manner most appropriate to the situation. In general, the more abstract and intellectual the required or expected linguistic reaction the less likely it is that the reaction or response will occur, In general also, responses that are expected to be externalized are likely to be more impaired than those which are personal and not intended for externalization, for reaction by a second individual. Put into simple language, aphasics can be expected to perform more appropriately than they can talk about their performance. It may be assumed, therefore, that the thinking associated with an individual or "private" symbol system is significantly less impaired than the externalized, productive language required to communicate thinking to another person.

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[1] Wilder Penfield and Lamar Roberts, Speech and Brain-Mechanisms (Princeton, New Jersey: Princeton University Press, 1959), p. 92.

Now this is a rather long definition of aphasia, but I don't know that I can give you a shorter one and still give you what I think is the essence of aphasia. I am going to proceed to give you a number of views of aphasia, some of which are still held rather dearly and some of which are dearly held at considerable cost to the patient. Aphasia was early viewed as a subtractive disturbance. This was a very convenient way to look at aphasia. According to this view, depending upon pathology, specific functions are disturbed which impair the individual's ability to deal with particular areas of symbol ability and related areas of ability. This essentially is the localizationist's viewpoint. Damage to a particular area causes specific dysfunction, that is, dysfunction is specifically correlated with lesions in a given area. There is a more generalized dysfunction because some functions are intercorrelated. To the best of my knowledge, Neilsen still believes essentially in this particular point of view. In regard to therapy and rehabilitation, Neilsen held--and Neilsen still holds--that the aphasic recovers if part of the other hemisphere, which was originally the subordinate one, is able to assume control of the disturbed function. Essentially then, you have a re-addition of the function that was disturbed or subtracted. This may be an over simplification of the point of view, but this is my interpretation of Neilsen's position.

A second position views aphasia as rather specifically a linguistic disturbance more than a disturbance in symbolic formulation. There is considerable evidence that the aphasic patient has greater difficulty in the use of conventional linguistic symbols and conventional language than in his personal or individual linguistic symbols. That is, the aphasic is able to think in a private language or a highly individualized symbol system. When he needs to translate his own symbols to conventional cultural symbols, he finds himself in difficulty. The aphasic's overt performances are frequently much more appropriate than his language associated with the performances. In an interview, we may note that aphasics who pay more attention to what they say break down in their performances more severely than ones who seem to pay little attention to their words, and more to their nonverbal behavior. This behavior may be related to feedback systems. Some of us are able to do things and talk without being disturbed by the things we say. Wendell Johnson once described a group of women busily chatting while they were knitting. Johnson observed that women talk while they knit so that they can have something to think about while they are talking. Presumably their thinking is related to their knitting rather than to their talking. If they paid attention to what they said then they might drop stitches and they would be knitting aphasically, or at least paraphasically.

Another point of view of aphasia is that it is a generally destructive disturbance. This is the position that Herbert Birch holds. He views aphasia as an omnibus disorder, in which traumatic alterations and cortical circumstances modify the normal patterns of excitation. Expressive disturbances, according to Birch, arise from any alterated relationship between the motor speech projection region and the remainder of the cerebral cortex. And so, for Birch, the job in the retraining of an aphasic patient is to find out how to get the patient to reorganize himself so that normal patterns of excitation again become established. Birch thought he had the answer with an approach which involved auditory stimulation of aphasic patients by intensified masking noise. The noises were of such intensity and range that the patients were not able to respond to incidental background noises. Presumably, the intensity of the noise somehow stimulated the brain. Burch found that the aphasic patients who were "fed" intense noise were able to respond much more appropriately to a set of pictures and verbal directions than when they were not so stimulated. Unfortunately, other investigators have replicated Birch's experiment but did not find the same results. We may conclude, then, that for Birch's particular population and the particular set of circumstances in which he made his investigation, his results may be appropriate.

One other point of view, attributed to Goldstein particularly, is that aphasia is essentially a psychological disorder resulting from cortical damage. The essential nature of the disorder is an impairment producing a reduced ability of the patient to deal with abstractions and an increased tendency towards concretism. Language disturbances are manifestations of the underlying disorder. To Goldstein then, and to those who believe in Goldstein's position, the essence of aphasia is in a reduced ability to deal with abstractions. Language is abstraction. For aphasics, the most abstract language is the most impaired, and the least abstract language is the least impaired. If you appreciate this point of view, you may then understand both the verbal and nonverbal behavior of an aphasic patient. What are the implications for rehabilitation from this viewpoint? There are at least two: somehow the patient must be helped to reorient himself to an abstract attitude, or if this cannot be accomplished, you must accept the patient's limitation in regard to language and deal with him on a concrete basis. You must appreciate that now you have a patient who can deal only with the concrete, and that presumably this will be a chronic state of mind and of linguistic behavior.

At this point I would like to read to you a criticism of the implications of Goldstein's position on aphasia and the nature of abstraction. Roger Brown[2] in his fascinating book, Words and Things, has one chapter called "Progressions and Pathologies," in which he presents and then attacks the notion that in pathology you necessarily have deterioration from the abstract to the concrete. Says Brown:

> There are three progressions: the evolution of the species from animal to man, the historical development of contemporary languages, and the recurrent transformation of children into adults. The early stages of all three progressions have been called concrete and their later stages abstract. If even this much be true we have a psychological equivalent of biology's famous: "ontogeny recapitulates phylogeny." The psychological development of the individual duplicates stages in the development of his species.
>
> The generalization implied by these literatures has still greater scope. Both aphasia and schizophrenia have been called psychological

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[2] Roger Brown, Words and Things (Glencoe, Illinois: The Free Press, 1958), pp. 264-265.

regressions. The aphasic has lost brain tissue. This loss presumably carried with it some acquired psychological functions and so returns the victim to an earlier developmental stage. Freud classified schizophrenia (he called it dementia praecox) as a narcissistic neurosis. This means that the schizophrenic's libido is concentrated on himself; he has none available for object cathexis. In a Freudian metaphor the schizophrenic is like an amoeba that sends out no pseudopodia. Its substance cannot be teased out from its center. Freud believed that the infant libido was also narcissistic and, consequently, that the schizophrenic was regressed to infancy. Since the cognitive processes of both aphasic and schizophrenic have been called concrete the total picture is consistent. Psychological and linguistic development are from the concrete to the abstract and regressive illness returns the human adult to the more primitive level.

This, I think, is also the position Roman Jakobson held until recently, in his consideration of the nature of aphasia. Jakobson held that the aphasic returned to an infantile level in his language and his thinking, and that one was a manifestation of the other. There is, of course, more than a mere germ of truth in what Goldstein and others who believe in this point of view hold. There is more than a germ of truth in every one of the positions that I have presented; but it may be that a view of what is concrete is a biased view, and that a word that is usually concrete may not be intended as concrete by a particular patient. What we really have are ego-oriented words. Perhaps ego-oriented is equivalent to Freud's concept of the narcissistic, and perhaps it is not. It may be that to be narcissistic is to have a proper appreciation of one's self for one's worth. It may be this is what the aphasic finally develops in his reorganization of his patterns of behavior. He is more concerned with how he feels and how he thinks, and how he expresses his feelings and his thinking, than he is with someone else's understanding of how he feels and how he thinks. It may be that when we get a patient who reorganizes along ego-oriented lines, we have quite a different person from one who continues to be concerned with how we think about how he thinks. It may also be that if a special kind of relationship is established between a patient and a clinician, and the patient is concerned with how the clinician thinks, he reorganizes his aphasia in the light of the clinician's view of what he is supposed to be.

From my own point of view, I view aphasia as initially a disruptive process that takes place because of brain pathology. This disruptive process breaks down previously established habit patterns of language and of other forms of behavior. Following the initial breakdown of established habit patterns there is a reorganization of behavior. This presupposes a reorganization of the neurological mechanism. We are not at all certain how this reorganization takes place but there is no question that it must take place. I think that there are certain predictable ways that the individual reorganizes, and these predictable ways are not predictable for all aphasics but are predictable in the light of the individual's own past history. I believe that a patient is inclined to reorganize according to how he viewed himself as an individual before he becomes aphasic. This is not too surprising. We all try to do this sort of thing. Some succeed in this reorganization, some do not. Some, when they fail to succeed or fail to improve sufficiently, then begin to accept other persons' notions about how this reorganization should take place. Thus therapy may determine reorganization. Some patients are more obdurate and resistant and do not go along with any reorganization lines but their own. I think that to find out how a patient reorganizes we had best dig into his history, and we will find a number of factors that are related to his reorganization and his possible recovery. How did he respond to illness at any time of his life? What evidence was there about premorbid psychological involvements? What happened as a result of his impairment? What gains were there? Some patients will tell you that they rather enjoy the attention they can get only when they are patients. They may be rather amused at being patients; it is not altogether an unhappy state of being. How much ego-orientation is there in the process of recovery? Are we dealing with psychopaths? Are we dealing with an individual who had a rather unusual system of logic, of rationalizing, and projecting? Are we dealing with the kind of an individual that I was talking about only yesterday at Dr. Burr's home? We were talking about a soldier I met during my military service who regularly went over the hill and who spent much more time in the guardhouse than with his company in training. When I asked him why he went over the hill so regularly, he said: "I've got to go and keep an eye on the mother of my six children." "You mean," I said, "you've got to go over the hill to see your wife." He answered: "Oh no, I didn't say my wife, I said the mother of my six children." "You're not married?" "No, I'm not married." "And why," I asked naively, "aren't you married to the mother of your six children?" The soldier, much more sophisticated than I, explained: "Well, I'm a hard drinking man and frankly not much good, and the way I look at it any woman who would marry a man like me isn't much good either and I'll be damned if I'll marry that kind of a woman." You see how completely logical the position is, if you can accept a certain premise and go on from that premise. Such fellows do not make good patients; they do not recover particularly well, but they seem to have no need to recover because according to their view they are not sick in the first place. It is you who does not understand them. I recall one in military service who became aphasic. He had a rather strange kind of anomia. He regularly referred to his mother by the name of his dead dog and to the dog by his mother's name. He was surprisingly consistent in the inter-change of names. It happened so regularly that I do not think this was a coincidence or a paraphasic accident. The dynamics behind this anomia had to be investigated.

Because time is almost up, let me give you one other notion that I have about aphasics. Let me share a very recent experience with you. This past week I examined a male aphasic patient at the Veterans Hospital in Brooklyn. In the sub-test that had to do with naming I held up my hand with my palm facing him and asked: "What is this?" He shook his hand and he said: "Oh! stop!" I thought that was an interesting response. I didn't know whether "Oh! stop!" was an in-class response or an out-of-class response. But it was certainly an interesting response and I did not consider it wrong. A hand held as I did is used as a "stop" sign, if not a "stop" symbol I next placed my hand on my cheek and asked: "What do you call this?" The patient imitated my movement and said: "Oh, oh, it's thinking." I made a mental note to ask myself where would I have put that answer if I were testing him with Wepman's inventory. What do you do with this kind of an answer? How do you objectively evaluate and score such responses?

In summary, what is aphasia? It is what happens to the behavior of an individual when he incurs brain damage. The brain damage is almost always incurred in the left hemisphere if the patient is right handed. And it is also more than less likely to be in the left hemisphere of patients who are left handed. Aphasia is a modification of all behavior of patterns. It is especially a modification in the language behavior of the individual. It is most especially a modification in that form and content of language behavior that is used conventionally for inter-personal and communicative purposes in a given culture

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Questions:

Participant Query: In your definition am I right in assuming that you said in right-handed people the damage is more apt to be in the left hemisphere, and vice-versa?

Dr. Eisenson: No, I did not say and vice-versa. Ninety-nine per cent plus of right-handed patients who become aphasic have damage in the left hemisphere of the brain. From 50 to 60 per cent of left-handed patients who become aphasic also have damage in the left hemisphere of the brain. Therefore, the notion that aphasia results from contra-lateral brain damage does not hold. If you want to generalize from current evidence you might say that regardless of handedness the likelihood is that aphasia in adults occurs as a result of damage to the left cerebral hemisphere.

Participant Query: May we infer that the speech really comes from the left hemisphere whether a person is right-handed or left-handed?

Dr. Eisenson: Well, for left-handed people the evidence seems to be this: the majority of left-handed persons have what we refer to as dominance for language in the left hemisphere. The evidence also seems to indicate that they make better and quicker spontaneous recovery than do most right-handed aphasics. To some clinicians this suggests that the differential in dominance between the two hemispheres is not as great for left-handed persons as it is for the right-handed. Therefore, it is possible for left-handed persons to reorganize neurologically with the right hemisphere assuming language control. A related conjecture might be that for left-handed persons the right hemisphere was probably always in there working along with the left hemisphere as a more equal partner than is the case for right-handed persons. Contemporary notions of cerebral dominance need and deserve considerable time for discussion. One of the questions I insist my graduate students must ask when they talk about dominance is, dominance for what? Dominance for what function? Dominance is not unitary for all functions: it is not all in the left hemisphere. Neither is it all in the right hemisphere. Some of my own research seems to suggest the kind of language that McDonald Critchley refers to as "super-propositional language" may be controlled in the right hemisphere rather than the left hemisphere and, therefore, some of the high subtleties and some of high level abstractions--the kind you must deal with if you have to do Miller analogies--are more likely to be impaired with damage to the right hemisphere than damage to the left hemisphere. In the light of this, one can conjecture other levels of language functions. You can talk about sub-propositional in terms of Hughlings Jackson. The automatic language which Jackson hypothesized "moved from the left hemisphere to the right hemisphere." This is how Jackson explained the maintenance of sub-propositional language when damage took place to the left hemisphere. The reason that a person can still swear, and count, and sing, and recite things that he committed very thoroughly to memory is that language, once it becomes habitual or automatic, is no longer under control of the left hemisphere. The left hemisphere is the hemisphere for ongoing linguistic events but not for the automatic ones. This type of language has control moved from the left to the right. We can talk about this as one level of language; the sub-propositional. Another language level is the propositional or communicative language; the language that we use in situations that involve an assumption of responsibility on the part of the individual, an assumption also of the need to communicate something to someone else. In addition, there is an expectation of a response that is directly related to the communicative effort; to the specific language used. This is propositional language. Super-propositional language may be exemplified by the language that the mathematician engages in when he is thinking or possibly talking to another mathematician, or a physicist to another physicist. It is language so abstract that it is above and beyond expectancy that anybody who is not a resident of an ivory tower of abstraction should be expected to be able to understand. We may postulate that this is a third level of language, and it may be possible--as data suggests--that this is controlled by the right cerebral hemisphere. Critchley also believes this to be the situation. Critchley observed that persons with right brain damage use a great many words and a great many circumlocutions, but almost always good ones, to arrive at the expression of an idea. The right brain-damaged person tends to be over verbose, indirect, and almost evasive in the answering of a question. It is an evasion because he can't think through quickly and directly, and verbalize readily for common consumption. There are some new notions about cerebral dominance and cerebral function in a recent book published by the Johns Hopkins Press. It is called Inter-Hemispheric Relations and Cerebral Dominance.[3] In it you will find several hundred pages of very exciting reading on what we mean by cerebral dominance.

Participant Query: Some time in the past I heard you comment on the fact that, in effect, we do not have two hemispheres, but rather two separate brains. Would you elaborate on this?

Dr. Eisenson: Yes, this again is a matter of trying to clarify notions by presenting another concept. What do we mean by cerebral dominance? If we think that we necessarily have two hemispheres because they look alike and are attached by some tissue that keeps them together--because the skull can't be depended upon to do it alone--then we assume that we have two hemispheres of one brain. But another way of viewing the matter is that we have two brains, which in gross appearance are mirror images of one another. They are, however, two brains under one skull; each having some functions which are quite different and some which are related. Together these two brains, or two semi-hemispheres, make it possible for human beings to behave as such. In some experiments with monkeys whose brains were divided, split right down the middle, they have been trained to do two things simultaneously. One brain, or one semi-hemisphere, did not interfere with the activities of the other brain. This is in support of the notion that perhaps we have two brains in one skull. This offers us another concept about cerebral functioning.

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[3] Vernon B. Mountcastle (ed.), Inter-Hemispheric Relations and Cerebral Dominance (Baltimore, Maryland: Johns Hopkins Press, 1962).

Participant Query: At the beginning of your talk you quoted several aphasic persons' descriptions of their feelings, and throughout your lecture this morning you have been describing the behavior of aphasics. If we were not at an aphasia conference we could, perhaps, attribute some of these descriptions to people who stutter. Since you have described aphasia as being a pathology of the brain, whereas stuttering is widely held to be functional, what is your feeling about the relationship between stuttering and aphasia?

Dr. Eisenson: This is what I call feeding me a question about which I like to talk. I do not view most stuttering as being functional, if by functional we mean psychogenic. I think most stutterers stutter because they are born with brains and nervous systems that function for language (speech) a bit deviantly. Stutterers do not necessarily possess "bad" brains, but different brains that do not permit them to deal with productive language quite the same way as most of us are able to deal with such language. For me, stuttering is not a speech defect but a linguistic defect, a language dysfunction. I think the analogies between stuttering and aphasia are rather remarkable, especially if you accept my definition of stuttering. The analogies become so remarkable that I at least feel they cannot be merely coincidental. I can refer you to the Symposium on Stuttering[4] of which I happen to be the Editor as well as a contributor, in which I present my particular point of view. If you want a quick answer, however, it would be this. Those linguistic situations which cause a stutterer the most difficulty are the same ones that cause the aphasic the most difficulty. They are also the same ones that cause normal people the most difficulty; so the difficulty may be a matter of degree. The kind of language the stutterer is most easily able to produce is the kind of language that the aphasic can most easily produce. Observations that mothers regularly report about their stuttering children are: "He doesn't have any trouble singing, and he doesn't have trouble saying things when he's angry." "Teacher tells me that he can recite with other children." "He says his prayers well, especially after he learns them; he has trouble only when he's learning his prayers. Once he's learned his prayers he says them well." Well, if you want to generalize from these frequently made observations, you could say that stutterers have least difficulty with sub-propositional language. Aphasics also have least difficulty with sub-propositional language. Most human beings have least difficulty with sub-propositional language. If you put yourself to the test you can read the newspaper, and get most of the meaning out of the newspaper while you are reciting the alphabet or counting aloud. If counting is sufficiently automatic, and reciting the alphabet is sufficiently automatic, you can produce this verbal behavior and still pay a fair amount of attention to what you are reading and understand most of the content. There are parallels between the two dysfunctions, stuttering and aphasia, that I consider not purely coincidental. I do not think stuttering results from an imposed pathology on the brain. I think that it is the result of a brain that develops in ways that are anomolous. Given time, perhaps the stutterer's brain organizes itself along more conventional lines. But there is always the possibility that, under conditions of pressure, this organization breaks down and the speaker gives way to earlier inclinations. He becomes linguistically disorganized and speaks like a stutterer.

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[4] Jon Eisenson (ed.), Stuttering: A Symposium (New York: Harper and Row, 1958).

DIFFERENCES IN LANGUAGE DISORDERS OF CHILDREN AND ADULTS

William G. Hardy

I have been more than a little ambivalent about trying to figure out how to present this particular topic; feeling somewhat like a birthmark on a handsome face when you turn the question of the aphasic adult around to some permutations as to what might be wrong with children. I do not think it will do any of us any harm, however, to undertake to try a slightly different overview. I am interested in some of the linguistic aspects of our problems. I do not know whether you have taken time to look through perhaps 8 or 10 major works, dictionaries and textbooks, to see what gets written down about what this problem is and what it means. It becomes very diffuse and diverse; a definition is apt to become very assertive and self-limiting. For instance, here is the Oxford reference for aphasia: "The loss of faculty of speech as a result of cerebral affection." This is a good early 18th century reference that really does not convey very much meaning. One of the problems we have to meet regularly, in dealing with very young children and their parents, is to try to find ways and means of making some of our concepts understandable. Most people apparently think of these various disorders in language purely on the expressive side. I stress this right at the beginning, because of course it is the intake-problems that are the major ones in all problems of children who do not normally, naturally, and readily learn language meanings and ultimately learn to talk. I know of no exceptions to this. There are various kinds of problems in children, of course, that produce different forms of aberrant, deviant behavior which have expressive aspects; unfortunately we see evidence of apraxic-like disturbances with very young children, as well as with adults, and this simply serves to confuse the issues. By-and-large, I will hold with my generalization that I have neither seen nor heard of a young child who has these difficulties in learning and remembering language modes, who does not have more or less serious problems at the level of intake.

There is a question that is asked recurrently. It is usually rhetorical. How can a child be aphasic if he has never developed language comprehension?

This recurs in cyclic terms, like the visit of the locusts, and every 6 or 7 years somebody gets re-exercised about the matter. I would like, in our discussion this morning, to make this something more than a rhetorical question; treat it as a genuine interrogative, and see if we can work out some answers to it. One way, perhaps, in which to get rid of verbal slavery in these terms is to agree (and we refer primarily to the child's problem) to let the term "aphasia" relate as broadly as we wish to the general idea of a basic incapacity in language comprehension and use--and then go from that point to try to describe and demonstrate what these incapacities are. On these grounds, I am convinced we can all find some real differences between children and adults, and between children and children, and between adults and adults. We need to know quite precisely which modalities are working and which are not, and whether central nervous system interferences involve problems of sensory integration, of comprehension, of formulation, of spontaneous expression, and, indeed, even of imitative capacity. I believe we have all been working in about the same direction for a good many years at a kind of pre-habilitation level with the adult in trying to determine, as well as we can, the details of his capacities and limitations in all aspects of language.

For reasons that are fairly obvious, this becomes rather difficult to do (if, indeed, it can be done at all) with the young child who is in a pre-language state. And so at the pre-habilitative level, the descriptions of affected children usually result from a long-term procedure that is sometimes called "diagnostic teaching." This is a matter of working with each child through a structured (and preferably carefully modality controlled) learning situation, in order to appraise his capacities and limitations.

With this much of a definition in the background, I believe it can become eminently worthwhile to compare various problems of children and adults in several ways involving behavioral relations between the self and its environment--in terms of demonstrative neurophysiologic conditions and states, and in terms of linguistic similarities and differences. In apriori terms, it seems quite reasonable to inquire about an individual's status prior to the onset of his aphasic state. In an adult, one would want to know in quite considerable detail about his intelligence, his aptitudes his attitudes, his social life, work habits, work skills, and so forth. One of the errors we all fall into (sometimes perforce and sometimes from omission), is that in many settings where adults are worked with we do not really have the capacity to find out enough of these details before we start working. Surely this is true of the general approach of medical practice to the problem of the adult who is ultimately going to turn out to be an aphasic individual. Hospitals are not geared to chronic treatment; they are designed specifically for acute treatment. The point is that 72 hours or so after the onset of many vascular accidents, the internist knows whether or not the person is going to live. If he is going to live, and the prediction is pretty clear that he is going to be aphasic, he should be moved immediately out of a hospital designed for acute treatment to facilities that are designed to help give him what he needs; which now is not life but the capacity to live. One specialist in this field wrote that how an adult is likely to respond to the immediate effects of aphasic involvement will depend in large part on what kind of person he was before he became aphasic. Apparently we may expect to find quite general agreement that the pre-morbid history of the individual is usually the most powerful controlling factor of his aphasic status. But what about the young child who is not developing language comprehension, nor the capacity to formulate and use verbal symbols? Precisely the same kind of determinations need to be made for such a child, but at much different levels and in much different ways. We can really know nothing about the pre-morbid state of a child who has never demonstrated capacity to develop language comprehension and use. We can only inquire into genetic possibilities, into the details of prenatal events, of paranatal experiences, of developmental and behavioral stages--all of which may or may not plausibly have been contributory to his status as we see him. Diagnostic judgments usually have to be made in terms of our past experience with other children, and in terms of inclusion or exclusion relative to other kinds of configuration, behavior, and learning--which by definition are something different from an aphasic state; and herein lies a fruitful resource for utter confusion.

Occasionally, one hears a professional comment to the effect that the diagnosis of aphasia in childhood commonly involves, on the part of the diagnostic group, a circular argument based on negative findings. A given child is demonstratively not mentally subnormal, he is not deaf; he is not autistic; he is not otherwise schizophrenic; he is not psychosocially deprived; he is not fundamentally emotionally disturbed; ergo he is aphasic. This kind of approach need not be a circular argument. It is quite logical, particularly if the diagnostic group is capable of making some very specific determinations of impairments of functions with regard to habits of attention, which is the primary point of foreground-background differentiations in the major sensory modalities, and of sensory integrative aspects of dealing with rapidly incoming information. The temporal dislocations are probably the most important ones that serve as interfering agents in the brain's capacity to manage information; to store and recall immediate sensory experience and long-term general experience. All these aspects of the brain's use of its energy contribute greatly to the economics of language relating the self to its environment.

At a rather more practical level of observing what a child does, it seems clear that many children are baffled by the demands of any situation that we may appropriately call communicative. With due regard for the fact that each deviant child probably composes a class of one, there seems to be two general and quite different groups of these deviant children. One child reflects basic interferences with attention mechanisms, with inadvertent shifts between distractability and perseveration; two sides of the same coin. This one is prone to be either stimulus-bound or extremely fleeting, as the details of situations vary. He seems to learn best within very limited boundaries of both stimulus and attention. The other child, in the second grouping, is quite gregarious but he has fundamental difficulties in sensory integration, in memory-storage, and in recall. For both children, the experience of communicating is a very baffling one. Children in the first group, if it be a valid grouping, are quite unpredictable. They may commonly swing very rapidly from a kind of other-worldly indifference to what is going on (which is why so many of them look as if they might be autistic), over to the opposite extreme of a frenetic drive (which is why so many of them are occasionally called schizophrenic). Those in the second group are just plain baffled and confused, because they are always several steps back in time away from the ongoing, incoming information Both groups of children demonstrate a very sharp contrast with the deaf three-year-old who has no other problems than lack of auditory sensitivity He will have learned by age three a very considerable amount of language through vision and seeing, which the aphasic child does not This is a negative finding with a very positive implication, and it is one I commend to your attention as you undertake to make difficult differentiations of pre-school age children

There is considerable current discussion about whether the child in the first group, the one with basic disturbances in the brain mechanisms of attention, should be called brain-injured rather than aphasic. I do not particularly like the term "brain-injured" as a useful descriptive for most young children we see It is apt to be a misleading term It misleads the direction of thinking both in terms of cause, if this can be known, and in terms of prediction about the child If one has to get the term "brain" into this hyphenated structure, I would prefer "brain-different" to "brain-injured," because with so many of these children there is not one iota of evidence from the genetic background, prenatal background, or paranatal background of the kinds of events one normally assigns to injury. Their developmental lacks and confusions differ, and just as many of them may be ultimately assignable to errors--inborn errors of intrauterine chemistry--as to anything else that is involved in the picture This differentiation in use of brain reference becomes fairly important, when you try to think forward in broad, sociological terms about where to place a child; and who is going to work with him. There are many schools who will accept a bright little boy who has attention-mechanism-interferences, but who will not accept a "brain-injured" child; albeit they both may be the same child. This is word magic, to which we are apt to become devoted The basic difficulties of the children in the first group show in many other ways beyond limitations in the learning and remembering of language, because the fundamental mechanisms of all aspects of learning and psychosocial adjustment are involved. So that really the terminology is quite academic If ways and means can be found to help this little fellow learn, through or around his distractabilities, then he can learn language and use it as a tool for other learning And if the child in the second group, whose problem is commonly labelled sensory aphasia, cannot learn through auditory-visual integration, memory and recall, then he is not going to use language as a tool for other kinds of learning, regardless of how we "labelize" him at the onset. Who is to say which child is the true aphasic--in any sense that is useful to the children?

The situation with the aphasic adult is quite different, largely, perhaps, because of his pre-traumatic experience. He, too, may commonly be frustrated or euphoric. Short of a very global involvement, however, he knows something of what is expected of him in a communicative situation, but he cannot meet it; and this produces a more or less serious breakdown in relations between the self and the environment. The status of the young child is quite different, because he cannot know what is expected of him. This is part of the problem of his learning and his adjustment. He simply cannot read the signs, whether or not he can say anything about them. Consequently, the aphasic child may on occasion, seem to be quite profoundly deaf, or stupid, or schizoid, or emotionally disturbed. Indeed, in terms of the demands and confusions in most communicative situations, day by day, his responses or lack of responses may exhibit any of the symptoms of these conditions. That is why our labelling habits need to be checked rather carefully. Moreover, because the learning and recalling and use of appropriate symbolic behavior depend on sensory integration, attention, and memory (these are factors common to all modes of learning), specific language disorders in childhood may not only be mistaken for, but may also accompany, these other kinds of disorders--particularly disorders involving refinements of sensory recognition and integration; a sub-topic we shall expand a bit later. This is especially true of deafness in children and with at least some kinds of what is nowadays called specific reading disability, where the line between sensory and linguistic problems is very thin indeed. In practical terms, I do not believe that our children can be very well segmented with the famous "Occam's razor," which is what is happening very commonly in the literature. The obvious recourse is to try to find out what they can and cannot do, in controlled circumstances of learning.

As time goes on, more and more becomes known about what happens, in terms of the neurochemistry and neurophysiology of the sensory nervous system, relative to these real or apparent problems in comprehension and use of language. In this regard, as well, the adult picture seems much clearer than that of the young child. Through the past 130 years or so, there have been very extensive studies and detailed reports of the effects of brain lesions in adults. There have been developed general patterns of association in professional minds between various sites of lesions and various kinds of dysfunctions. Largely depending on whether a particular critical observer is fragmentistic or holistic in his view of the functioning person, various schools of thought have had their opponents and their many followers. Rather more recently, largely at the level of rehabilitative practicality, there has been developed the thesis that what is needed is a detailed description of the impairment of function relative to residual function; that is, what the person can and cannot do in communication in terms of linguistic comprehension and use Through all of the confusion of current debate about adult aphasia there seems to run at least one thread of agreement. Nobody seems to expect the injured adult brain to be able to develop a new pattern of performance. There may be a considerable measure of spontaneous recovery of function, which means simply that neural tissue and networks controlling these functions are not permanently obliterated; but there cannot, in reasonable probability, be an[illegible]newal or replacement of neural tissue or of the controlling neural networks. [illegible]he adult brain is set in its patterns of habitual responses to familiar stimuli. Part of the diagnostic task in adult aphasia--possibly the most important part, once we know the person is going to live (and we must know that before we can know he is aphasic)--is the careful description of the individual's range of accomplishments and limitations, particularly of the brain's residual linguistic tools.

The situation with the very young child is quite different. His brain in early life is plastic. It is highly resistant to further insult, even after profound trauma. There has not been a single recorded instance of a child, age three or less, who has become permanently aphasic by way of traumatic brain damage of the sort that commonly causes adult aphasia. If he lives, provided that his development was reasonably normal up to the time of whatever happened, he can be expected to go on to learn language comprehension and use.

What of the so-called "congenitally aphasic child?" His brain is presumably equally plastic; albeit interferred with, in the sense that various neural networks may not be working in normal ways. The diagnostic need remains the same; to try to delineate the tools of his successes and failures, relative to the brain's means for learning language comprehension and use. This is commonly a difficult task, and thus the need for differentiation from other diagnostic entities with symptoms which (at any given moment in time) may be quite similar to those of an aphasic problem. It has been emphasized that these differentiations can usually be made only by ruling out such basic factors as mental subnormality or autism, and by exposing the young child who does not demonstrate a capacity for language comprehension to highly structured, modality-centered diagnostic teaching. There are proponents of the idea that the very young child without the capacity of language is, to all intents and purposes, schizophrenic. If this means that he is in a state of communicative confusion, so be it; the use of the term "schizophrenic" adds very little to the comprehension of his problem. If it means something else; that, for instance, he is forever bereft of the capacity to use verbal stimulation as a mediating representation of habitual responses to linguistic stimuli, then this use of the term "schizophrenia" suggests some special pleading, which only substitutes a generalization to fill the lack of the required description of specific impairments. Current findings in the laboratory, the clinic, and in pre-school training situations all indicate that this young brain is indeed plastic and capable of learning, through or around many kinds of interferences. It remains for all of us to learn better how to determine with each child what the interferences are, so that the unimpaired modalities may be used for learning the substance of language.

In this regard again (and now we are at the third point in measuring linguistic potentials and lacks), there are some basic differences between the problems of children and adults. Assuming that an aphasic adult has led a reasonably normal life up to the time of the traumatic insult, one may work out an intricate scheme for appraising his capacities and limitations. Hildred Schuell and her colleagues have over 700 items in their Minnesota Test battery. Joseph Wepman has emphasized the need to determine the basic controlling modalities prior to the aphasic status. Our friend, Dr. Eisenson, has stressed the importance of the pre-morbid personality traits of the patient. Schuell emphasizes the diversity of what she calls preceptive effects, and the importance of feedback in broad elaborations of the individual's use of language in relating himself with his environment. She calls attention to extrinsic areas of the brain, involving large tracts of afferent inflow in the projection areas, and to intrinsic, interacting connections, parts of which, at least, are modality-specific. You will find very extensive development of this last idea in some of the reports from Karl Pribram of his work at Stanford. Schuell's five major classifications of adult aphasia--ranging from the simple to the global--are based specifically on patterns of impairment, the findings from her test-battery, and she stresses the point that there is no need for concern with degrees of severity within the classifications. The accomplishment of re-learning is a function of the nature of the problem, not of the severity of the problem. The basis for these classifications is the determination of the amount of available language in all modalities, with specific measurements of auditory understanding, reading, spelling, writing, and arithmetic; together with levels of any apparent perceptual or motor deficits. It is readily apparent that few of these data are accessible in evaluation of the young child who does not demonstrate language comprehension and use.

Wepman, on the other hand, finds useful analogies between normal developmental stages of language in childhood and various stages or types of aphasic problems of the adult. In his thinking, the infantile status of pre-speech is related to global aphasia; and the babbling-cooing developmental stage to the jargonic manifestations in the adult. The third level, which a couple of weeks ago, at least, he called fortuitous speech (and by which I think he means something like Vigotsky's description of the infantile verbal gesture) in childhood development suggests pragmatic aphasia. Fourth, we see the infant's beginning use of substantive symbols, albeit in a limited fashion, which may be correlated with semantic aphasia. Finally, the development of grammatical sequence in the child relates to the adult problems of syntactic aphasia. One can be quite certain it is not by chance that Wepman's last three types--pragmatic, semantic, and syntatic--represent the basic categories of semiosis (the study of meaning) and the philosophy of C. S. Peirce who was Chicago's greatest bulwark of modern pragmatism. In practical terms, as one observes the very young child's communicative status, this classification is useful to the extent that the observer can determine the child's access to the various sensory modalities, in terms of integration, formulation, and expression.

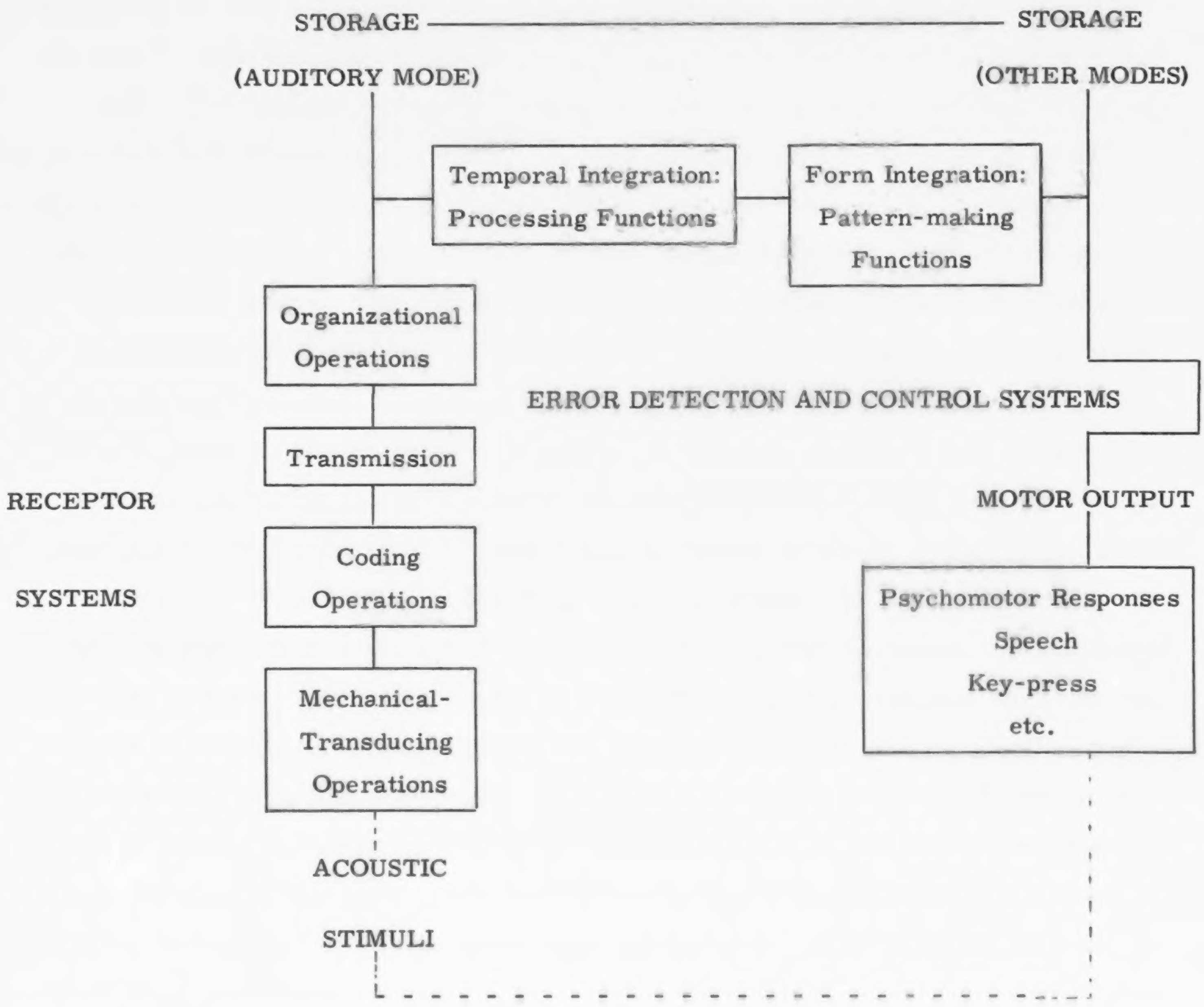
A nonverbal child without apparent motor deficiencies may ultimately fall within any of the first three of Schuell's five classifications. In the early years he may either seem to be global or jargonic, in Wepman's classification. But this does not mean that any given child will remain linguistically impaired in any of these ways. This is the sharpest point of difference with adults. In adulthood, with due regard to pre-morbid status but now relative only to the brain's linguistic functions, it seems to make very little difference whether one is dealing with a very young person, a middle aged person, or an aged person. The nature and quality of affect may be observed with considerable accuracy, and I think, with some high degree of predictability. With the young child, the little chap with the plastic brain, this is not so. Children fall into other categories. One finds oneself using phrases like "He has no language comprehension at all," or "He has a little in some modalities and none in others," or "He has some, but he doesn't have enough anywhere." This is the kind of qualification that usually creeps into the description. The trouble, of course, is that many details of learning of any kind are relatable to the apparent malfunctions which are commonly generalized as childhood aphasia. These include attention, storage, recall, auding, vising, tacting, and habituating in terms of responses to linguistic signs. Lacks in any or all of these capacities may be involved in what is called "childhood aphasia," with or without any perceptual or motor problem.

Many experts in the management of subjects with aphasia agree that quite commonly there are interferences with the detail of informational intake, relative to the usual requirements of the communicative situation. Let us examine what might be the nature of this interference--in terms of one sensory modality and the brain's use of this modality. Sensory aphasia in childhood these days is becoming a very familiar reference. In several ways this is quite a peculiar term, for it apparently represents a confusion of the sensation and the brain's use of the sensation, one being information-bearing and the other being information-using.

How the brain deals with sound is a fascinating study. In mammals approaching the level of man, there are many projection areas and there is no place in brain tissue, cortical or sub-cortical, where electrical potentials may not be elicited by introducing sound as the stimulus. Responses can be elicited from every major knowable and describable anatomical structure of the mid-brain, the inter-brain, the entire sub-cortical structure, and all the loops that connect both hemispheres of the brain. Even with children, therefore, we are not always dealing with a site-specific kind of thing, and I am not at all sure that we are dealing commonly with a site-specific problem in adults. This is one of the reasons why simple, routine audiometry is rarely sufficient in trying to appraise either the child's problems or the adult's problems; beyond the point of demonstrating that he has some access to sound. Three of the functions of the auditory system are: sensitivity, discrimination, and recognition. What gets confused in the adult because of vascular accident, trauma, or tumor, and in the child, because of a whole galaxy of etiologies (of which we know very little), is not simply the sensitivity-bearing function of the auditory system, but the discriminatory and the recognitive functions of the system. The function of sensitivity is a basic auditory function, and it must be adequate for anything else to happen; but for us to be able to use auditory information we must be able to discriminate, and we must be able to recognize refinements relative to temporal event-patterns of frequency, intensity, and rate of change. The idea that deafness means only loss of sensitivity is absurd. This is only one aspect of impairment of the hearing mechanism, and in the problems we are discussing this week is by no means the most important one. A given aphasic person may also have some presbycusis, but this need not have any direct relationship to the aphasic state; it is just an accompanying source of psychosocial confusion. What we need to learn better to do (and several groups are now working quite intensely on this), is to find out more about the details of the kind of temporal integration that the intricate cortical levels of the auditory mechanism are capable or not capable of performing. The only technique that I know for this (and it is strictly in the experimental stage), is to work directly with durational measurements concerned with the relations between kind and length of signal and intensity of signal. One is not dealing here with the problem of detection, but with the problem of recognition relative to sensory detail. At stake here is the brain's summating capacity to utilize from bilateral input the kinds of auditory information we all take for granted. In this regard, we are still talking about VIIIth Nerve functions.

The functions of the integrating systems are: processing, pattern-making, and the usual time-binder, retention, which makes a feedback system possible in the management of rapidly incoming acoustic information. I do not believe that the concepts of processing and pattern-making are materially different from each other. One leads to the other. What our brains have to do relative to the information that our auditory mechanisms present to the brain is to manage very rapidly incoming bits of information. It makes little difference whether you analyze the nature of these bits in terms of phonetic detail or in terms of phonemic, meaningful understanding. Again in linguistic terms, we expect patterns to emerge from these bits and pieces of information. Yet, as you think of the dispersion of this information at the cortical level, it really becomes a rather fantastic study to figure out how any of us has ever been able to put to use, and to learn from, these bits and pieces that compose the aggregate we talk about in terms of pitch and loudness at the psychoacoustic level of hearing and listening. Our brains must use all these bits and pieces "as a whole," and somehow remember what has happened before, and be able to relate it to what is happening now, and then be able to develop a pattern from it to help formulate what is going to happen next. All of which are the important temporal aspects of linguistic comprehension and use. The following schema indicates some of this in a more organized fashion.

C N S CONTROL OF SENSORY INFORMATION IN RELATIONS BETWEEN ACOUSTIC STIMULI AND PSYCHOMOTOR RESPONSES


STORAGE
(AUDITORY MODE)
STORAGE
(OTHER MODES)
Temporal Integration: Processing Functions
Form Integration: Pattern-making Functions
Organizational Operations
ERROR DETECTION AND CONTROL SYSTEMS
Transmission
RECEPTOR SYSTEMS
MOTOR OUTPUT
Coding Operations
Psychomotor Responses
Speech
Key-press
etc.
Mechanical-Transducing Operations
ACOUSTIC STIMULI

The boxes on the left side of the schema represent ways to refer to (not to describe) the organization of the VIIIth Nerve system. Mechanical transducing operations include what goes on in the external and middle ears, and probably what goes on in the inner ear structure--in the fluid wave-train of the hydraulic system of the inner ear, at least up to the level of the shearing action of the hair cells. This seems to be the last step, short of chemical change, that succeeds in transducing and producing electrical current. There is reason to suppose that there may be some chemical interferences present in the cochlear duct, quite as mechanically interfering with auditory detection as some of the more obvious problems that we can describe in the middle ear. The last job that is done--somewhere between the hair cells and the ganglion cells--is to create electrical energy which is coded in the system. If you try to read the code, the only way you can pick it off is the familiar N^1 - N^2 scheme. If you put in a pure tone, what you are going to see on the oscilloscope (usually with an electrode pickup from the round window membrane), is a very faithful reproduction of that signal; that is, not yet coded information. Then, past the immediate coding operation is the transmission series, involving many primary synaptic junctions. The conductivity of the long axons is an all-or-none function. But a great deal can happen--to interfere and change and modify and modulate and add and subtract --at each one of the major synaptic junctions. The neuro-synapses in this system, as is true in most sensory systems, serve in effect as generators. They recreate, if you will, the possibility for coded information to go into a next series of conductivity. Conductivity either occurs or does not occur, but a great many changes can occur at the synaptic levels. There are at least three major ones in the brain stem. There is one rising up through the lateral lemniscus; there is another major one at the level of the colliculi; and a principle, multiple, complex synaptic series at the medial geniculate level in the thalamus. Some of the signals are conducted around the brachium of the colliculi below; some of them go through the brachium, but they all apparently center in the medial geniculate. Yet, there is a case on record of a young man we happen to have known, an aphasic child, who went through quite a good developmental course in a special curriculum in a special institution. By the time he was 9 years old, about 25 people had managed to demonstrate at least 50 hearing test well within normal range; he had a little speech, and he could actually give back some heard speech-signals as well. Then, he was killed in an accident. When his brain was sectioned, it was found that he had no medial geniculate bodies on either side. There was no substance of neural structure which could possibly have conveyed the ascending afferent auditory system. We do not know what he did all the hearing with, but he did hear and he was using his hearing. We do not know very much about many of these problems, once we leave the end organ.

Then, we get up to the organizational operations. (I have already referred to what I think are the most important aspects of these: how the brain serves in differentiating, summarizing, and discriminating at the level of refined recognition with bits and pieces of acoustic information.) Next, we have a storage mode relative to this sensory experience, and then we move out of the main auditory system. Now, we are probably everywhere in the brain, in terms of integration in time relative to incoming information. First, the brain lines up the information and develops some patterns from it, of one sort or another. Then, we must have and must use access to other kinds of storage modes, in that many of our current experiences have some relation to previous ones. Ultimately, according to the nature of the situation, we have an appropriate psychomotor response. We are at a sub-linguistic level here. One can readily set up an experiment wherein your verbal response will be relative to nonsense syllables, or wherein you are asked only to press a key according to the kind of auditory stimulus presented to you. There need be no relation to meaning. I believe that one can explain every test of audition in terms of that kind of loop.

In a very intensive study, with a select group of children, we are concentrating on some of the correlates that seem to introduce difficulty in terms of continuing attention centering and memory, storage and recall, relative to these rapidly incoming bits and pieces of information. We are not now talking about classical deafness; these are not problems of sensitivity. One of the things that slows up our non-language-learning-comprehending children is that they do not have access to redundancy. Each experience, even the simple auditory one, may remain quite unique. None of us could do well in learning language under those circumstances. This is where auding comes in. Auding is what we are doing when we are working with central integration of acoustic information in terms of processing and pattern-making. This is the brain's management of what is being presented. It is what we all mean, I believe, when we talk about understanding what we hear; but we first have to hear it. Auding is what none of us does very well, when after two or three years of college courses in a foreign language we go abroad where the language is native and try to converse with some of the natives. We may know a considerable vocabulary; we may be quite fluent in reading it, but it comes so fast in conversation we just cannot keep up with it. We cannot sort it out. I believe this is an excellent analogy in terms of these temporal problems of processing and pattern-making. After you live in the country for a while, if you have a reasonable "ear" and are interested in all of this, what happens? You begin to understand what is being said. Your hearing has not changed a bit but you are auding more efficiently. The lack of this capacity for auding is what is wrong with many children who are labelled "sensory aphasics." This is one of the primary breakdowns. There are many possible ways to account for it in neurophysiologic, neurochemical, and neuroanatomic terms. We are beginning to see some laboratory evidence, under carefully controlled conditions in work with animals, of concepts and states that relate to what some of our mixed up children do, or do not do. (See Windle's article in Science, June 13, 1963.) We simply do not know much as yet (neuroanatomically, neurophysiologically, or neurochemically) about most of these brain activities, to which we are so acutely alerted in terms of anybody's aphasic problems--child or adult.

LINGUISTIC CHANGES IN APHASIC SPEECH

Harold Goodglass

The purpose of this presentation is to discuss the effects of aphasia on the linguistic features of speech. The pursuit of this problem has brought us back repeatedly to investigations of normal adult language and studies of language development in children. Hopefully, it will also help provide a link between clinical descriptions of aphasic abnormalities and the physiological substrate related to the given complaint.

What do we refer to by the term linguistic features and what does linguistic science have to contribute in this area? The concept, as I use it, includes all the vocabulary elements and the rules for combining them, by which a given language can be completely described. Linguistic features are properties of the language, as it is used--not of the speaker. Thus, the expressive and receptive aspects of a linguistic variable are not distinguished. We who study aphasia, however, are concerned with the speaker's behavior, with respect to the linguistic features of his communication. Therefore our techniques and subject matter can best be classified as psycholinguistic. For example, from the point of view of linguistics, it is significant to distinguish between voiced and unvoiced consonants in English; once this distinction is recognized as significant, it applies to the production and the comprehension of the distinction, equally. However, from the psycholinguistic viewpoint, we ask whether the auditory comprehension of the distinction is a prerequisite for its correct use by an aphasic in his speech.

The identity of expressive and receptive linguistic features does not extend from the auditory-vocal modality to the modalities of written language. The rules for describing a spoken language are only partially represented in the written version, and only at certain points is written language isomorphic with the spoken. The fact that our written language is roughly phonetic heightens our illusion that it is based on entirely the same rules as speech. The most familiar points of difference are in the conventions of spelling. For example homophones like hoes and hose can be distinguished in writing but not in speech, while the words use (verb) and use (noun) can be distinguished in speech, but not writing.

Now we are approaching the answer to the question of what the linguist can contribute. Most of us have at our command an elementary or high school version of the linguistic rules of our own language. We believe in the existence of parts of speech, called "nouns," "pronouns," "articles," "verbs," and so forth. We believe in the differences between complete sentences and non-sentences, and we may even have learned some of the phonetic categories that are significant in English. However, when it comes to studying the stress and intonational features of speech, we are immediately aware of our total lack of ready made categories and our lack of training in listening to these features of language. But in other features of speech, as well as intonation, the modern linguist has observed, classified, and theorized about regularities of our language which we are only dimly aware of using. He assesses their psychological significance in terms of their presence in other families of languages. Modern linguistics for example is dealing with theories of sentence generation, which have direct implications for the study of grammatical disturbances in speech.

The traditional content of an aphasia examination gives no clue to the significance of linguistic changes. The traditional aphasia examination attempts to measure the impairment of language performance in each of the various modalities of input and response: namely, speech, auditory comprehension, reading, and writing. Various combinations of input and response modalities are usually also tested. For example, taking auditory comprehension as the input channel, one tests response by repetition, by writing, by pointing to a named object, and so forth. With some refinement in the examination, the expressive modalities may be further broken down to distinguish the motor-skill aspect (articulation) from the symbol evocation aspect (word finding).

If the manifestations of aphasia were adequately accounted for by the profile of comparative impairment in the various input and response modalities, the topic of linguistic changes would be superfluous. The fact, however, is that some of the most dramatic differences among types of aphasics are completely missed by measurements of the modalities. These differences relate to symptoms which have seemed to demand linguistic terms in order to describe them terms like "anomia," or the loss of nouns; "dysprosody" or the disturbance of intonational patterns; "agrammatism" and "paragrammatism." In this fact lies a double challenge, for if nature has in different individuals experimentally removed the capacity for carrying out certain classes of linguistic operations, we have:

1) The possibility of checking out theories as to the organization of these linguistic functions and their relative difficulty.

2) The possibility of reducing the linguistic description to terms which fit the existing body of neurophysiological and psychological theory and knowledge. Experience has taught us that the terms with which we classify a symptom may have no relation to the psychological change which accounts for it. Aphasia abounds with descriptive terms for various symptoms; terms which imply a theoretical framework, but which in fact have only a pragmatic descriptive value.

To review some of the linguistically defined symptoms, and theories about them, we can list:

The phenomenon of anomia. The observation repeatedly made by Jackson, Goldstein, Head, Weisenburg, and McBride, is that the production of nouns seems to be separable from the remainder of language This would mean that the semantic processes can be isolated neurophysiologically from the syntactic processes. The fact that severe isolated naming difficulties have consistently been related to destruction of tissue in the posterior temporal or temporo-parietal regions adds circumstantial evidence in favor of the hypothesis that nature has made a special provision for the operation of naming things However, some recent investigations (particularly those by Howes and Geschwind and by Wepman's group) imply that it is not necessarily the case that naming is organized as a qualitatively different type of process, that it is only in a quantitative sense that it differs from the stringing of words into familiar sequences. Both of these research groups have considered an explanation based primarily on the factor of frequency of word usage. I use this issue to illustrate the fact that the logic by which we analyze language performances may result in categories which have no counterpart in nature and, in fact, may prevent us from seeing what is happening.

The problem of agrammatism. A most challenging linguistic feature in aphasia is the existence of a group of patients whose symptomatology appears to be the reverse of what has been described for the anomic. These patients may have little difficulty in naming objects, but have conspicuous difficulty in putting words into sentences. Since this speech pattern is characteristically associated with awkwardness of motor speech production, it has been called motor agrammatism. The usual listing of the component elements of agrammatic speech reflects an assumption that an underlying impairment of a grammatical nature is responsible. For example, the agrammatic aphasic is said to have lost the use of the grammatical function-words and particles; such as articles, prepositions, inflectional endings. The patient's own self analysis tends to make the examiner see the defect in grammatical terms, for it is familiar to hear a patient say something like: "By, From, With--I don't know--all mixed up." The motor agrammatic also sounds to us as though his repertory of sentence types is restricted to the simplest, shortest and most commonly occurring in everyday speech.

There are some unexplored rough edges around the case for a generalized aphasia of grammar. One is in the uncertain relationship between expression and comprehension of grammatical distinction. In some constructions, it really does appear that the patient no longer understands or notices the difference between contrasting grammatical forms, such as verb tense inflections. Thus, his failure to make the distinction in his speech is then taken as evidence that there is a basic capacity for using grammatical relationships, which is subject to injury in certain forms of aphasia. However, we find that more often the typical motor agrammatic understands very well the distinctions which he does not express in own speech. On the other hand the fluent-speaking aphasic may spontaneously use, in perfectly correct form, a construction which he cannot distinguish when he is required to listen to it.

Some years ago, the linguist Roman Jakobson, brushing aside all these petty inconsistencies, proposed a very attractive dichotomy of aphasia into cases of what he called "contiguity disorder" and "similarity disorder." These terms correspond to idealized descriptions of motor agrammatism on one hand and anomia on the other. While Jakobson's dichotomy can be criticized for prematurely imposing a loose philosophical unity on a complicated collection of symptoms, it had the advantage of dramatizing this striking problem in aphasia and illustrating the interest of the linguistic community in the theoretical challenge of aphasia phenomena.

I have expressed some apprehension earlier about the danger of reifying the name of a symptom by assuming the name to have explanatory value. Nevertheless, our research strategy has been to assume that the symptom as defined is indeed a first approximation to an explanation of the process which produces it. Thus, in our study, we have taken each of the features attributed to motor agrammatism and attempted to measure them in highly structured test situations, using sentence comprehension, repetition and sentence manipulation techniques.

Rather than list the various experiments that have been done, I would like now to sketch the point of view towards which we have been pushed by our evidence, and refer to the evidence in the course of this discussion. Our studies have concentrated on the problem of motor agrammatism, and it is in this area that we believe we have made some progress.

As it now appears to us, the distinguishing characteristics of motor agrammatism arise from the difficulty these patients experience in initiating and maintaining the production of a sequence of speech sounds. Here we are offering an essentially sublinguistic explanation to account for the specific grammatical difficulties which are unique to this form of aphasia. In order to take this position, however, we have to postulate a variable which we name the "psychological saliency" of words. Saliency is defined as the resultant of the informational value, affective value, and phonological prominence of the word as it occurs in the message. The motor aphasic with agrammatism is a patient who needs a salient word in order to initiate an utterance; he can maintain the flow of speech for relatively few syllables. He then must find a new salient word for another start. Since most grammatical function words are low in salience, they are lost from the opening of sentences and often from the middle. The patient gravitates to the salient word, and having broken into speech at the wrong part of the sentence, he is saddled with an "agrammatic" utterance.

This interpretation of motor agrammatism is not too different, at first glance, from what has been written before. Isserlin, for example, stated that the motor agrammatic, because it is difficult for him to produce speech, has an urgency to deal only with the most essential words; he therefore condenses his speech to telegraphic form. I have even been given this explanation by a patient for his own telegraphic speech, but I cannot accept it. We see too much evidence of these patients struggling to grammatize their speech by means of some formula or other. It cannot be simply a need to condense and get it out before it escapes.

As for the emergence of salient words--Bonnhoeffer, the German neurologist, has already said that the function words are more difficult to produce because of their low meaning-value.

I wish to point out that neither Bonnhoeffer's nor Isserlin's explanations would have predicted a greater difficulty with unstressed function words in the first position. The explanation I have offered leads to the expectation that the function word will not be lost so often when it immediately follows the stressed or salient word. It also leads to the expectation that function words which normally carry a stress in the initial position may well be spared.

Our evidence is the following: first, the objective measure which most strikingly agreed with clinical classification of agrammatics, was the length of uninterrupted word sequences in free conversation. The speech of the agrammatics is so heavily weighted with one and two word units, separated from each other by hesitation and pauses, that this feature forces itself into the definition of the agrammatic speech pattern. Our second finding led us to doubt that the concepts of grammar are any more impaired in the so-called motor agrammatic than they are in the fluent speaking amnesic or Wernicke aphasic of similar functional disability On examining the order of difficulty of the various English inflectional endings and the order of difficulty for our repetition of sentences of different grammatical types, we find the same sequence of difficulty in both groups. It is true that the agrammatic makes more errors, but both groups are vulnerable to the same grammatical complexities.

The conspicuously heavy drop out of grammatical function words in the first position of sentences was in line with the need of the short-phrase dominant aphasic for a salient word to begin an utterance. The long-phrase dominant patients actually had more omissions of salient words at the beginning of sentences. I have mentioned the prediction that the same function words would be retained if they immediately followed a stressed opening word, but lost if they preceded the stressed word. This prediction was dramatically verified in the first patient who came on the scene with a classical case of agrammatic speech.

Our most severe agrammatics have been patients who also had a very short memory span for repetition of words--no more than three or four isolated words reproduced at a time. It is hard to see how such a patient could project the plan for a complex sentence, even if he could speak word by word. The rule of span for word repetition is a factor to be considered. If a knowledge of grammar is not specifically impaired in the case of the so-called motor agrammatic--or, in other words, if the fluent-sounding Wernicke aphasic is equally disturbed in his awareness of grammatical relationships--how is it that this type of aphasic may be so fluent in the production of syntactic forms? I would suggest that the fluency of these patients does indeed cover an equal amount of confusion in grammatical concepts as exists in the motor aphasic. The difference is that the sensory aphasic or the anomic aphasic have not suffered a disturbance in the automatisms of producing motor speech sequences. In particular, it has been suggested that the first aspect of a sentence arising in the normal speaker's mind is the intonational and rhythmic pattern that corresponds to the general intent of the sentence to be spoken. The actual syntactic sequences of words are presumably closely conditioned to the aroused melodic pattern.

When, as in motor agrammatism, these automatisms are disrupted, we see the patients struggling to apply consciously his residual knowledge of the rules of grammar. This effort usually results in stereotyped misuse of a few simple constructions. Sheer frequency of usage in his past life seems to determine what form the patient's error will take. For example, the present progressive of the verb, which is probably the most common form of the verb in everyday speech, occurs repeatedly as an incorrect form in the speech of motor aphasics.

We are currently comparing the ability to discriminate grammatical distinctions when presented auditorily with the ability to use the same distinctions in speech. In this experiment and in studies of inflectional forms we have found ourselves closely paralleling some of the techniques used in the investigation of speech acquisition in children.

In our investigations, we have dealt only incidentally with the problem of naming disorder. Our evidence indicates that this does indeed represent a specific defect which is demonstrably more of a problem in the non-agrammatic patient than in the agrammatic. An analysis of the naming process, in a way that sheds more light on the various forms of naming disorder in aphasia, is an essential contribution to the linguistic nature of aphasic disorders at this point.

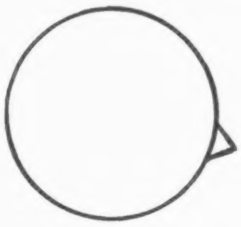
INFORMATION THEORY MODELS AND APHASIA

William E. Castle

Though information theory developed primarily out of concerns for more economical means of transmitting telegraphic and telephonic signals, the principles of this theory have often been applied to the broad area of communication disorders. This paper is an attempt to relate your interests in aphasia to some basic models from information theory. It is hoped that this can be accomplished by describing these models in some detail and demonstrating how they have been used in the disciplines known as psycholinguistics and linguistics for discussing aphasia.

The theorists who talk about the measurement of information are interested in finding a way to measure the physical characteristics of any kind of signal used for transmitting messages from one point to another. Hopefully, there can be a common measure for all kinds of such signals. For the most part, these theorists arrive at their objective by using mathematical models. This paper is not a discussion of these mathematical models but rather of some simple analogues to these models.

Perhaps the model most basic to all information measurement could be depicted in a manner such as that shown here in Figure 1. This model can be referred to as the binary choice model.



ON = (presence of energy flow) = "yes" = 1 = +

OFF = (absence of energy flow) = "no" = 0 = -

Figure 1. The binary choice model.

The figure presented is that of an on-off switch of the type used in electric circuitry. This on-off switch is used to determine whether there shall be a flow of current or not. The choice that can be made by the operator of the switch is two-fold, or binary. These choices can be said to be translatable into a code which is strictly binary in nature; that is, there is flow of energy or there is not; yes or no; 1 or 0; plus or minus.

Assuming that the signs for message construction can be restricted to two, the next thing to be considered, according to Miller,[1] and according to Weaver,[2] is the fact that the amount of information involved in any mode of communication is a function of the number of alternative messages available for message construction. Thus, if the possible messages each have only one sign in its make-up, there is the possibility of choosing between two messages, 0 and 1 (if such be the particular binary symbology chosen for use). If the possible messages each has two signs, however, the choices will be four in number: 00, 01, 10, and 11. If each possible message has three signs, there would be eight such messages: 000, 001, 010, 011, 100, 101, 110, and 111 and so on. The number of choices of message increases logarithmically as the number of signs in each message increases linearly. On the basis of this relationship, Shannon and Weaver[3] and Fano[4] have defined information mathematically as $\log_2 N$. In this formula, N stands for the number of alternative messages and the base 2 logarithm represents the binary choice model that has been described. Any resultant of the formula is described in terms of bits, a word which is short for binary digit and which is used to describe the number of binary digits required to create each alternative message ideally possible in a given communication mode. For instance, in the regular English alphabet, there are 26 characters. If each character is considered an alternative message, it might be said that each character then grants $\log_2 26$, or about 4.56 bits of information. This means that ideally a secondary alphabet of 26 messages should be creatable in which each message is made up of 4.56 binary digits (0's and 1's). Mathematically this is feasible. In the world of practicalities, it is not quite possible; but the method of measurement is, nevertheless, exemplified.

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[1] G. A. Miller, Language and Communication (New York: McGraw-Hill Book Co., Inc., 1951).

[2] C. E. Shannon and W. Weaver, The Mathematical Theory of Communication (Urbana: University of Illinois Press, 1949).

[3] Ibid.

[4] R. M. Fano, "The Information Theory Point of View in Speech Communication," Journal of Acoustical Society of America, XXII (1950), 691-696.

A second basic model from information theory is one which has been presented by Shannon and Weaver.[5] It is depicted in Figure 2. This model might be called the communication system model, since it has probable usefulness for describing any communication system. At the information source some message is selected for transmission. In speech the information source is the brain of the speaker; in telegraphy, the source is any number of combinations of dih and dah; in telephony, it is the signal transmitted to the diaphragm of the mouthpiece. The transmitter in the system always changes the selected message into some signal form; in speech, the transmitter would be the vocal mechanism, which produces varying sound pressures through time; in telegraphy, the transmitter is the telegraph key that interrupts a current of electricity; and in telephony, it is the immediate device that changes sound pressures into varying electrical current. Thus some signal is created to be sent over a channel. In speech, the usual channel is the air medium; in telegraphy and telephony, it is usually wire. The receiver is an inverse transmitter, so to speak, and it changes the signal back into some form of message, which may or may not be interpretable, but which should be measurable for amount of information. Ideally, the amount of information, in bits, at this point should be the same as the amount of information presented to the transmitter. But, because this is seldom the case, the factor of noise is provided for in the model. Noise is taken to mean any element of distortion that creates an addition to or subtraction from the signal's physical information value as it travels across a given channel, or transmission line.

Interpretation of the model shown in Figure 2 is sufficiently loose to allow for operationally defining the transmitter of the system to be any one of a number of things in the process of transferring speech signals to a specific listener: (1) it may be the speaker's vocal mechanism; (2) it may be the tympanic membrane of the hearer and the ossicular chain to which it attaches; (3) it may also be the stapes, which sets up mechanical activity in the fluids of the inner ear; (4) it could be the hair cells, that, hypothetically at least, transform the mechanical activity of the inner ear fluids into that type of electrical activity that makes up nerve impulses; and (5) it could be any one of a number of specific points within the make-up of the highly complex nervous system. In effect, almost any element in the intricate system of speech communication may be called either transmitter or receiver, depending on the point of reference.

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[5] Shannon and Weaver, op. cit.

Still a third basic model from information theory needs to be described. In Figure 3 is shown a diagrammatic representation[6] of that situation in which anyone finds himself whenever he drives an automobile. Literally, the word automobile means self-moving; but in most respects the word is a misnomer. At present, at least, the motion of automobiles must be initiated, directed, and stopped by some outside force, the driver. On the other hand, although the driver may transmit orders to the automobile which start, speed up, slow down, or stop the motor or which make the total vehicle turn right, turn left, go forward, go backward, or stop; these orders are governed in large part by the actions of the automobile. In other words, the driver and the automobile regulate and inform one another. They are involved in a feedback loop, the simple analogue for servomechanisms or control systems. The third basic model may thus be referred to as the feedback model.

These three basic models have been used in the realms of psycholinguistics and linguistics to help describe or explain aphasia. For instance, Figure 4 shows a diagram which is a complex of binary alternatives, simple communication systems, and feedback loops. This diagram was presented in 1960 by Wepman, Jones, Bock, and Van Pelt[7] as an operational description of three levels of language function in man's central nervous system.

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[6] G. T. Guilbaud, What is Cybernetics? (New York: Grove Press, 1960).

[7] J. M. Wepman, L. V. Jones, R. D. Bock, and D. Van Pelt, "Studies in Aphasia: Background and Theoretical Formulations," Journal of Speech and Hearing Disorders, XXV (November, 1960), 326.

For best understanding the diagram in Figure 4, let it be assumed that the central nervous system is a highly complex information processing system. The input to the central nervous system is described in the lower left corner of the diagram. Most forms of sensory information enter the system here by being translated into sensory nerve impulses. Wepman _et al_. only describe the auditory and visual forms of sensory information. Throughout the diagram the auditory and visual information modalities are paralleled with one another by pairs of arrows which designate the alternative lines along which the informations might be transmitted.

Initially, any sensory nerve impulses based on auditory or visual stimuli may alternatively be held at the lowest of the three levels of language function described (the _reflex_ level) or be directed to the next level above (the _perceptual_ level) This is the most immediate exemplification in Figure 4 of the binary choice model which has been described. If the sensory impulses are held at the reflex level, they may or may not travel the transmission line that leads to the reflex level of the central nervous system. At this point, the sensory impulses may or may not be translated into motor nerve impulses. Finally, the motor impulses may or may not be transmitted to the ultimate output stage of the system, where they may or may not prompt motor activity. Within the limits of the lowest level of language function described in Figure 4, the binary choice model operates repeatedly in several successive stages. At the same time, the simple communication model is represented over and over again by a series of transmitter-channel-receiver combinations, in any one of which noises might interfere with transmission. It will be apparent that both the binary choice and the communication system models operate successively at a similar rate throughout all stages or levels of the system being described. From this point on, therefore, attention will not be specifically drawn to this fact.

If input stimuli travel all the way across the lowest transmission system in the diagram shown in Figure 4 and are, at the output stages, transformed into motor acts, _reflex_ behaviors are said to have occurred. The authors of the diagram describe these reflexive behaviors operationally as the lowest level of language function in the central nervous system. As such, these behaviors are distinguished from the other two levels of language function shown in two ways. First, they are said to have no obvious connection with the so-called memory-bank, or the recall function. Second, the reflex behaviors are "not usually conceived as part of the language mechanism" at all.[8] They are given such consideration by Wepman et al. because of the differential diagnostic import they have to neurologists for describing the relative intactness of neural structures. Perhaps the operational description here should be carried one step further so that the reflex level of language function is secondarily described as the level of no language.

If the sensory nerve impulses are shunted to the perceptual level shown in Figure 4, they may alternatively be held at this level or be directed to the next level above (the conceptual level). If they remain at the perceptual level, they may or may not be reflected to the memory-bank either for storage or for comparison to previously stored information. They may or may not be transmitted to the appropriate central mechanisms. Within the central mechanisms, they may or may not be translated into a complex of motor impulse messages that are based on previously learned patterns.

If the input stimuli travel all the way across the second-level transmission system shown in Figure 4 and are, in the final translation stages, transformed into motor acts, imitative behaviors are said to have occurred. The authors of the diagram describe this perceptual-imitative level of language function operationally as that level which leaves its traces on the memory-bank but which has no meaning to the individual involved. Such behavior is exemplified by the child who produces echoic language, by persons who repeat words from a foreign language but do not know the meanings of the words, and by aphasic patients who can copy figures that they see without understanding the meaning of the figures.

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[8] Ibid., p. 327.

If the sensory nerve impulses are shunted to the conceptual level, then they may or may not be transmitted to the central mechanisms appropriate to this level. Within these central mechanisms they may or may not be used through integration for concept formations. If they are used to form concepts through integration, theoretically this is done in three steps. First, meaning is attached to whatever sensory information has reached the central mechanisms by associating this information with that information which is stored in the memory bank; this is called the *associative* process. Next, the sensory information is transformed into conventional symbols (appropriate word patterns) from the individual's language; this is called the *semantic* process. Finally, the selected symbols are put together in sequences (grammatical forms), appropriate to the individual's language; this is called the *syntactic* process. The concepts which are formed may then either be stored in the memory-bank for later recall or be translated immediately into a complex of motor impulse messages based on previous experience.

If the input stimuli travel all the way across the third-level transmission system of Figure 4 and are, in the final translation stages, transformed into motor acts, *expressive* language behaviors are said to have occurred. These may take the forms of speech, writing, or gesture. Wepman *et al*. describe this third level of language function operationally as that level which not only leaves its traces on the memory-bank but which also is meaningful to the individual. Such behavior is optimally exemplified by that which is called normal language behavior in the human being.

The diagram in Figure 4 was conceived primarily in an effort to better explain behaviors which occur in adult aphasics. Wepman *et al*. consider *aphasia* to be any disturbance of the processes in the central nervous system used for integrating input stimuli with previously learned language patterns. Those persons who cannot relate the input stimulus to their language are said to have *pragmatic* aphasia, those who have trouble with the selection or formation of meaningful symbols (words) are said to have *semantic* aphasia; and those whose grammar and syntax are incorrect or missing are said to have *syntactic* aphasia. It is implied in Figure 4 that Wepman *et al*. believe disturbances that occur at any point in the system other than within the integrating mechanisms are defects (noises) in transmission and should be named differently. Such disturbances on the input side of the system are labelled agnosias and those on the output side are labelled apraxias.

There is still one other matter, expressed briefly in Figure 4, which demands specific attention. This is the matter of feedback. The diagram from Wepman et al. merely alludes to external feedback, but cognizance is given by these authors to the importance of both internal and external feedback, particularly in rehabilitative techniques for aphasics which are based on restimulation and self-correction.

Disruption of the feedback process apparently has its most important role in effects on the output, or expressive, language functions. This apparent fact has been illustrated with considerable precision by Fairbanks,[9] who has conceptualized one of the expressive language modalities, speech, as an elaborate servosystem of the sort shown in Figure 5.

Let it be assumed that some single-word concept has been selected from the memory-bank of Figure 4 and directed into the storage area of the controller unit, designated in Figure 5, for the speaking process. Successive units of instruction about how this concept should be translated into a spoken word are transmitted simultaneously to a mixer and to a comparator. The successive instructional units become successive control points for the processing of the speech signal by also serving as successive unit goals for the output of the effector unit.

The effector unit for speech, which consists of a motor (the respiratory system), a generator (the vocal folds) and a modulator (the resonation-articulation structures) is directed toward each successive unit goal of the output by an appropriate complex of motor nerve impulses. As this is happening, the sensor unit continually monitors the mechanical operation of the effector unit and continually relays data about that operation back to the controller unit, in the form of feedback signals. Three sensors participate in the activity of the sensor unit. These are sensor 1, the auditory mechanisms, which receive information by either channel 1 (the air) or channel 2 (the body tissue); sensor 2, the tactile sense organs; and sensor 3, the proprioceptive end-organs.

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[9] G. Fairbanks, "Systematic Research in Experimental Phonetics: 1. A Theory of the Speech Mechanism as a Servosystem," Journal of Speech and Hearing Disorders, XIX (June, 1954), 136.

The various feedback signals produced by the sensor unit are sent to the comparator. Here they are compared with the input signal for the mixer, and an error signal is continually calculated which indicates at any given time "the amount by which the intended speech unit, then displayed in the storage device, has not yet been produced by the effector."[10] The error signal is also fed into the mixer, where it may be used to modify the unit of instruction to the effector unit, by way of the effective driving signal, in such a way that error is brought to zero, or as near to zero as possible.

Systems similar to that devised by Fairbanks for the speech modality may also be conceived for the writing and gesture expressive modalities. Each would show, in its own way, a similar complex of feedback loops.

Obviously, a decrease in auditory, visual, tactile, or proprioceptive feedback guidance to expressive behaviors as a result of damage to the central nervous system is a matter of considerable importance. An aphasic patient who has what Wepman et al. would call auditory agnosia, for example, cannot monitor his oral behavior auditorily and, therefore, cannot guide his expression as readily. An aphasic patient with some neurological impairment of vision will have difficulty maintaining accuracy of both his writing and his reading.

In the realm of linguistics per se a method for talking about levels of language function is often used, which may be just a detailed expansion of the conceptual level purported by Wepman et al., but which is, nevertheless, also interesting in itself. The diagram in Figure 6, suggested by Weinreich,[11] is a representative linguistic description of how language is perceived and produced. In this diagram, the highest functional language unit is labelled understanding. According to Weinreich, understanding involves a stored vocabulary of meaningful items, against which are checked the grammar analyses during input (decoding) of language and from which are selected the items used for grammar analyses during output (encoding) of language. In this sense, the understanding and grammar levels are like the memory-bank and the syntactic process, respectively, as proposed by Wepman et al. The phoneme (speech sound) analyzers and the grapheme (the alphabet character) analyzers are apparently considered to be the first stages of input analysis. Certain linguists might argue that the functional units of grapheme and phoneme should be replaced, as the smallest unit of input and/or output, by what is operationally called the syllable. Others might argue that in between the lowest levels and the grammar level should be inserted, perhaps with broken-line borders, an intermediating level--often referred to in linguistic theory as the morpheme level. Be that as it may, the diagram proposed by Weinreich is a sufficient take-off point for summarizing two hypotheses about aphasia which have been proposed by Roman Jakobson, a linguist of considerable renown.

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[10] Ibid., p. 137.

[11] C. E. Osgood and M. S. Miron, (eds.), Approaches to the Study of Aphasia (Urbana: University of Illinois Press, 1963).

For both the phoneme-grapheme level and the grammar level of language, Jakobson describes two dimensions: the paradigmatic dimension and the syntagmatic dimension.[12] The first refers to selection of the appropriate linguistic symbols in a given context, and the second refers to selection of the appropriate sequencing of linguistic symbols in a given context. For a context such as "The man hit the _____," normal syntagmatic mechanisms for English decide that some noun must fill the blank, but the paradigmatic mechanisms for English decide which noun must fill the blank. For a context such as "b___t," normal syntagmatic mechanisms for English, perhaps, decide that some vowel phoneme or grapheme must fill the blank, but the paradigmatic mechanisms for English decide which vowel phoneme or grapheme must fill the blank.

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[12] R. Jakobson and M. Halle, Fundamentals of Language ('S-Gravenhage: Mouton and Company, 1956).

Jakobson has hypothesized that two extreme types of aphasia exist, each of which ties itself to one or the other of the paradigmatic and syntagmatic dimensions of language at the level of grammar. Patients with defective paradigmatic mechanisms on the grammar level have what Jakobson calls a similarity disorder. Their chief difficulties are in word-finding, labelling, categorizing, and initiating utterances that contain nominal terms; this aphasia syndrome seems to be the same as what Wepman calls semantic aphasia. On the other hand, patients with defective syntagmatic mechanisms on the grammar level have what Jakobson calls a contiguity disorder. Their chief difficulties are in combining words into phrases or sentences and making transformations from one grammatical voice to another, from one tense to another, and so on. This aphasia syndrome is probably the same as what Wepman calls syntactic aphasia.

Before Jakobson's second proposal a propos to aphasia is introduced and summarized, it might be appropriate to discuss for a moment the distinctive feature theory, suggested by Jakobson, Fant, and Halle,[13] for how the phonemes of a given language are distinguished from one another. This brief discussion is offered not only as a background for Jakobson's other suggestion about aphasia, but it exemplifies once more the usefulness of the binary choice model described earlier.

In essence, this theory suggests that at a level of linguistic consideration lower than the phoneme, each phoneme can be said to be made up of a bundle of distinctive features which serves to distinguish that phoneme from all others in the same language. In Figure 7 is shown a description of nine distinctive features, proposed by Jakobson et al. in 1952[14] as those pertinent to English. Note that each of the nine features is described in a binary manner: vocalic versus non-vocalic, consonantal versus non-consonantal, compact versus diffuse, and so on. Notice also that in the table shown in Figure 7, the symbology used is binary: the plus sign is used to indicate whether the positive element of the feature (the left-hand member) is relevant to a given phoneme; the minus sign is used to indicate when the negative element (the right-hand member) of the feature is relevant.

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[13] R. Jakobson, C. G. M. Fant, and M. Halle, Preliminaries to Speech Analysis: The Distinctive Features and Their Correlates (Cambridge: The M. I. T. Press, 1961).

[14] Ibid., p. 43.

Some time before the distinctive feature theory was described, Jakobson had proposed that loss of phoneme distinctions has a regularity which mirrors, in its order, the pattern of acquisition of phoneme distinctions in children.[15] By implication, the aphasic should be trained for reacquisition of these distinctions in accordance with the acquisition pattern of the child. This order has been described in the book entitled Fundamentals of Language, published by Mouton and Co. in 1956.[16]

The evidence available, which cannot be reviewed in this paper, appears not to support this latter proposal from Jakobson. On the other hand, a fair amount of clinical evidence appears to bear out his similarity-contiguity dichotomy of aphasia disorder. It remains for future research to establish full validity for either of these hypotheses as well as for the specific descriptions reviewed herein from Wepman et al., Fairbanks, and Weinreich. All of these descriptions have served well, however, to illustrate what usefulness the binary choice, the communication system, and the feedback models have for discussing aphasia.

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[15] Osgood and Miron, op. cit.

[16] Jakobson and Halle, op. cit.

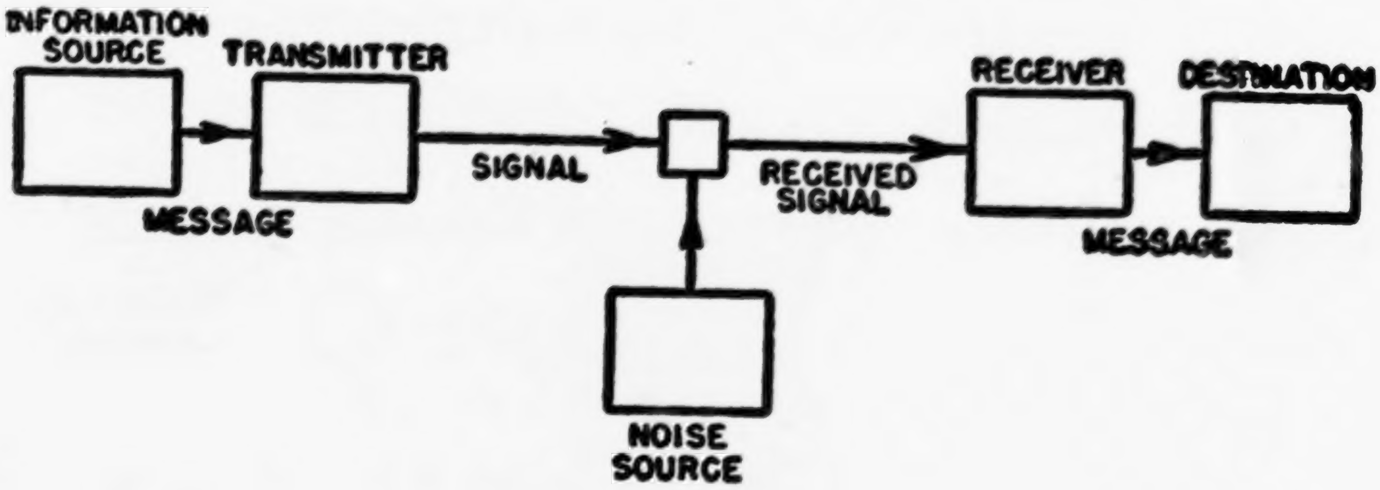

INFORMATION SOURCE
TRANSMITTER
RECEIVER
DESTINATION
SIGNAL
RECEIVED SIGNAL
MESSAGE
MESSAGE
NOISE SOURCE

Figure 2. A communication system model. Reproduced with permission from Shannon and Weaver[2].

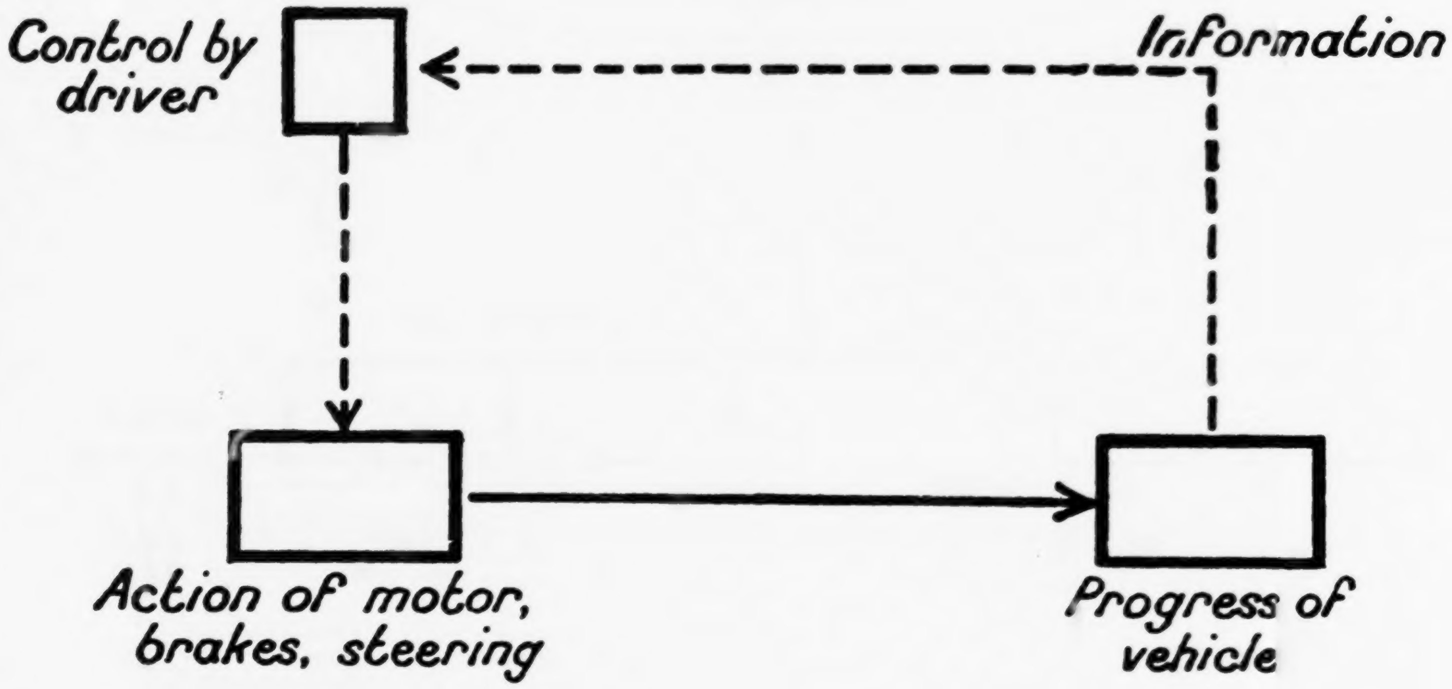
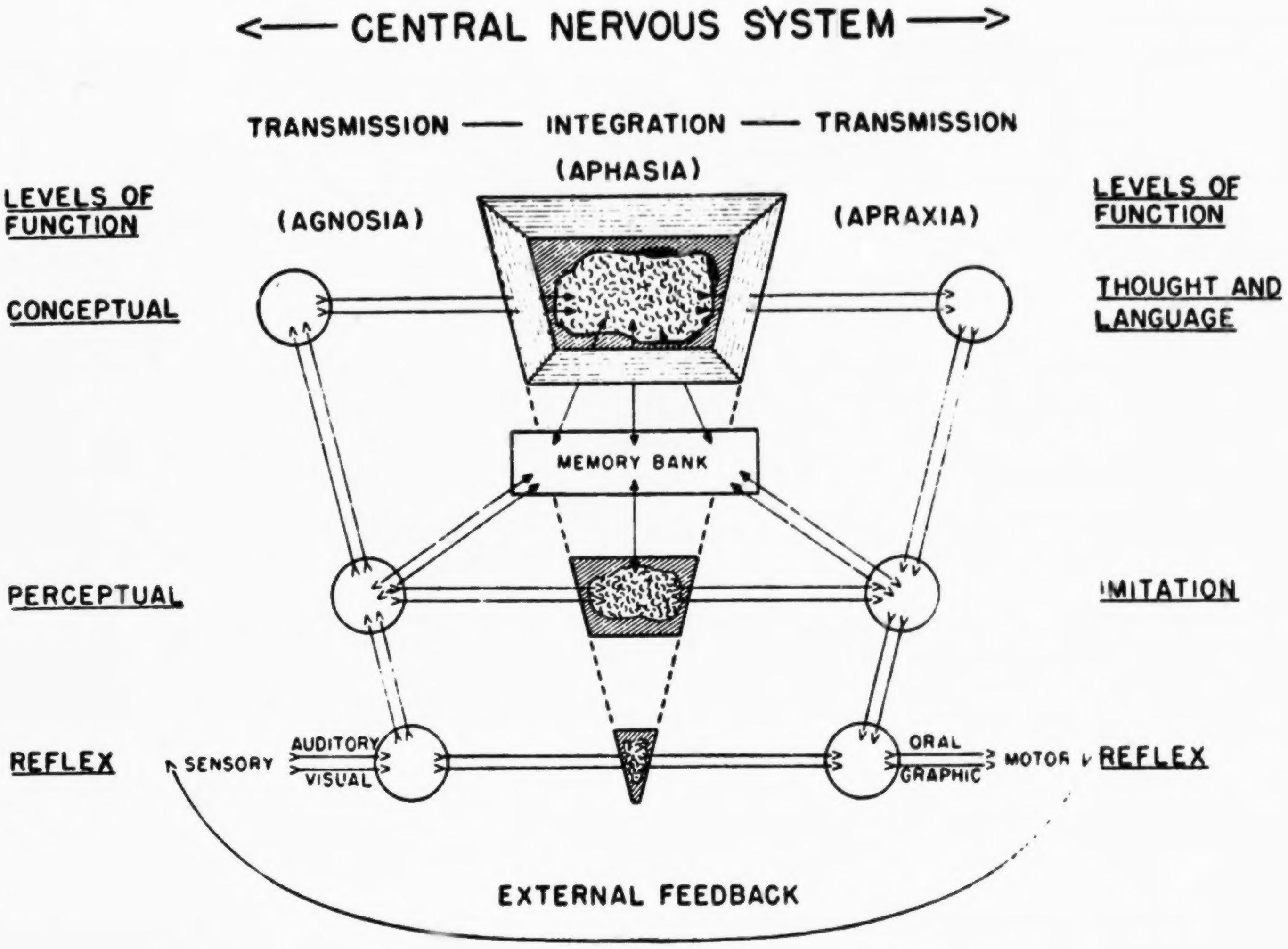
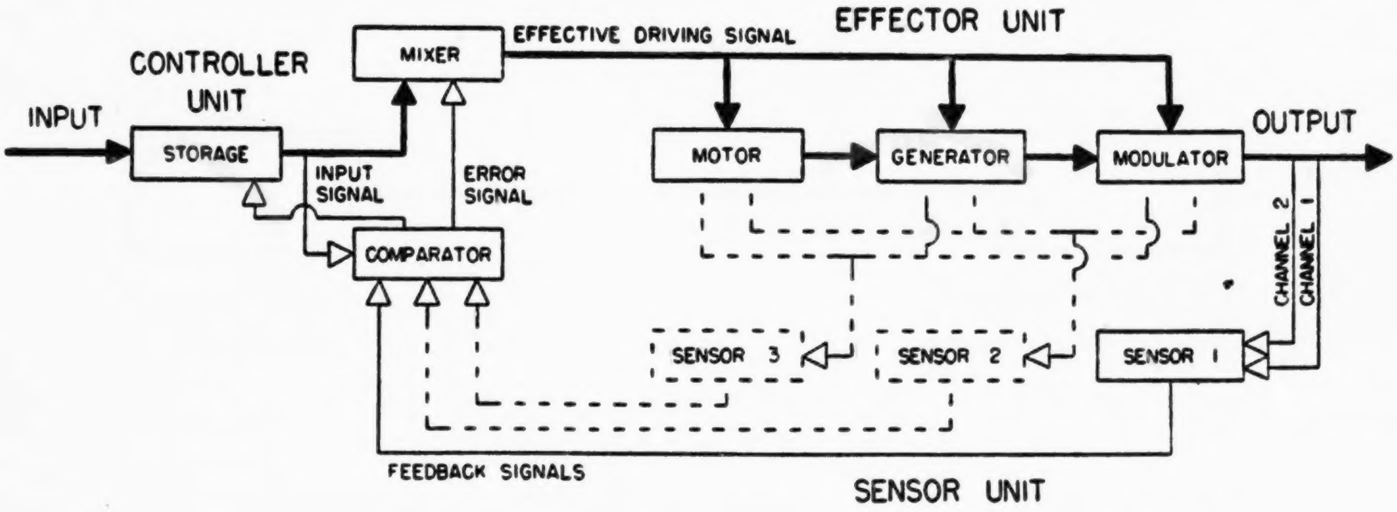

Control by driver
Information
Action of motor, brakes, steering
Progress of vehicle

Figure 3. A feedback model. Reproduced with permission from Guilbaud[6].



<—— CENTRAL NERVOUS SYSTEM ——>
TRANSMISSION —— INTEGRATION —— TRANSMISSION
(APHASIA)
LEVELS OF FUNCTION
(AGNOSIA)
(APRAXIA)
LEVELS OF FUNCTION
CONCEPTUAL
THOUGHT AND LANGUAGE
MEMORY BANK
PERCEPTUAL
IMITATION
REFLEX
SENSORY
AUDITORY
VISUAL
ORAL
GRAPHIC
MOTOR
REFLEX
EXTERNAL FEEDBACK

Figure 4. An operational diagram of the levels of function in the CNS. Reproduced with permission from Wepman, Jones, Bock, and Van Pelt[7].



INPUT
CONTROLLER UNIT
STORAGE
MIXER
EFFECTIVE DRIVING SIGNAL
EFFECTOR UNIT
INPUT SIGNAL
ERROR SIGNAL
COMPARATOR
MOTOR
GENERATOR
MODULATOR
OUTPUT
CHANNEL 2
CHANNEL 1
SENSOR 3
SENSOR 2
SENSOR 1
FEEDBACK SIGNALS
SENSOR UNIT

Figure 5. A model of a closed cycle control system for speaking. Reproduced with permission from Fairbanks[9].

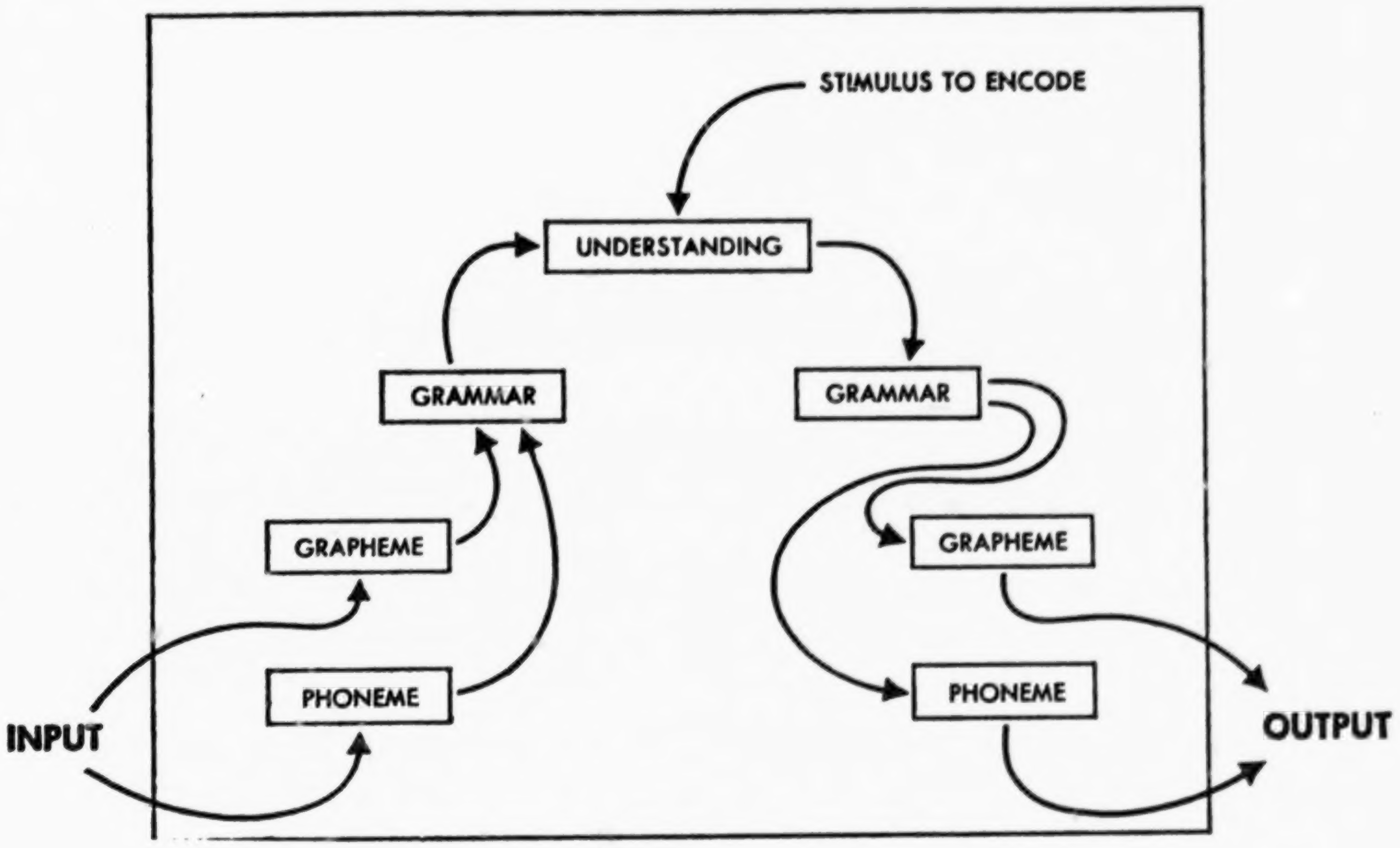

STIMULUS TO ENCODE
UNDERSTANDING
GRAMMAR
GRAMMAR
GRAPHEME
GRAPHEME
PHONEME
PHONEME
INPUT
OUTPUT

Figure 6. A functional model of language behavior as proposed by Weinreich. Reproduced with permission from Osgood and Miron[11].

	o	a	e	u	ə	i	l	ŋ	ʃ	ʃ̂	k	ʒ	ʒ̂	g	m	f	p	v	b	n	s	θ	t	z	ð	d	h	#
1. Vocalic/Non-vocalic	+	+	+	+	+	+	+	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−
2. Consonantal/Non-consonantal	−	−	−	−	−	−	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	−	−
3. Compact/Diffuse	+	+	+	−	−	−		+	+	+	+	+	+	+	−	−	−	−	−	−	−	−	−	−	−	−		
4. Grave/Acute	+	+	−	+	+	−									+	+	+	+	+	−	−	−	−	−	−	−		
5. Flat/Plain	+	−		+	−																							
6. Nasal/Oral								+	−	−	−	−	−	−	+	−	−	−	−	+	−	−	−	−	−	−		
7. Tense/Lax									+	+	+	−	−	−		+	+	−	−		+	+	+	−	−	−	+	−
8. Continuant/Interrupted									+	−	−	+	−	−		+	−	+	−		+	+	−	+	+	−		
9. Strident/Mellow										+	−		+	−							+	−		+	−			

Figure 7. The pattern of distinctive features for the phonemes of English proposed by Jakobson, Fant, and Halle. Reproduced with permission from Preliminaries to Speech Analysis[13]

HISTORICAL PERSPECTIVE: PIONEERS IN APHASIA

Walther Reise

The literal meaning of aphasia is loss of power of speech. The name "aphasia" was given to it by Trousseau (1868) with the intention to substitute a linguistically irreproachable term for the linguistically misleading term of "aphemia," chosen originally by Broca (1861). In purely descriptive terms, aphasia is disordered language resulting from brain lesions. In historical terms, it comprises: (1) the first unmistakable description of speech disturbances in brain lesions; a clinical subject, and (2) the first attempt to correlate these disturbances with a well-defined region of the brain; an anatomical subject. It is impossible in an historical investigation of our subject to isolate these various aspects from one another.

Though cases of aphasia might have been known to antiquity, and though writers of the sixteenth and eighteenth centuries described loss of speech, which was interpreted by writers of the nineteenth century as true aphasia, none of these ancient and more or less forgotten early authors could be called pioneers in aphasia. Their descriptions remained unnoticed; they left no traces in the history of aphasia; they did not prepare the way for others. The pioneers in aphasia emerged in the nineteenth century. It is no accident that the first elaboration and diagnostic refinement of aphasia took place in the nineteenth century which, after an initial vitalistic stage, was anxious to sponsor and to save a concept of science free from speculation and based on faithful observation. The informed student of the history of medical ideas will not be surprised to discover one of the first and best descriptions of a case of amnesic aphasia (or nominal defect) in Pinel's Medico-Philosophical Treatise on Mental Alienation (1809). Pinel, who has been called for more than one reason the founder of modern psychiatry, tried to revive and to take advantage of the observational principles of Hippocratic medicine.

Approximately a hundred years ago, in April 1861, the Paris Anthropological Society witnessed Paul Broca's demonstration of the brain of a 51 year old patient, who had been affected by a right hemiplegia, and who had been unable to speak more than the single syllable "tan," for 21 years. He repeated this one syllable endlessly and used it as his only answer to any question. To be exact, I should add, he also had one other word, a curse, which he used in anger. The disorder of speech was accompanied by an incomplete preservation of understanding of spoken language and of communication through gestures and mimicry. Broca demonstrated a rather extensive softening of the left hemisphere of the brain. He considered it a justifiable assumption that the oldest and most advanced part of the lesion was in the left frontal lobe. He concluded that the frontal lesion was the cause of the loss of speech. In other words, he limited himself to a localization of symptoms, avoiding a localization of functions. This caution was also expressed in the title of his communication: "Loss of Speech, Chronic Softening and Partial Destruction of the Left Anterior Lobe of the Brain."

In August of the same year, in the Paris Anatomical Society, Broca again seized upon the opportunity to discuss this subject. At the outset of his communication, he presented Bouillaud's theory of the site of speech, or rather of articulatory power, as confirming his own observation. Bouillaud, Broca said, had saved from a shipwrecked phrenology a site of speech founded on pathology.

He called this disorder "aphemia" and explained it by a central disorder of coordination; a locomotor ataxia of articulatory movements. Though he did not intend to maintain that each elementary function has its seat in a circumscribed cerebral convolution, he still believed that large areas of the brain correspond to large areas of the mind. But this is a captious and ambiguous conclusion, in which the word and the concept of area are used at times with a psychical and at other times with a physical (spatial) connotation. Above all, the title of his communication was now different, as follows: "Notes on the Seat of the Faculty of Articulate Language Followed by an Observation on Aphemia." Therefore, from April to August, 1861, Broca made the decisive and ominous shift from a localization of symptoms to a localization of functions. It is true that, in spite of the title, he still reached at the end of his second communication the following, still cautious, formula "The integrity of the third (and perhaps also of the second) frontal convolution seems to be indispensable for the performance of the function of articulate speech."

In 1864, about three years after his first communication Broca delivered a lecture to the Paris Surgical Society. In this lecture, he pronounced his final localization of aphemia, which he correlated with lesions of the posterior third of the third frontal convolution of the left hemisphere. He was able to refer to two observations of traumatic aphemia, made by Ange Duval, in which speech disorders were used successfully for the first time in regional diagnosis during life. Broca ascribed the value of a successful experiment to these observations; in both instances, conditions of investigation were ideal in view of the strictly localized and isolated lesions. At the same meeting he insisted that the left hemisphere always has to be involved in order to produce aphemia. He considered this fact to be "strange," without being able to explain it. Obviously, he did not as yet consider any relationship to handedness. At the end of his lecture to the Paris Surgical Society, Broca again acknowledged a localization of functions as follows: "The faculty of articulated language is localized in the left hemisphere of the brain or, at least . . . it depends mainly on that hemisphere." As far as I can see this was Broca's final view.

At the October 5, 1865 meeting of the Paris Anthropological Society, Broca supported the view expressed with caution by Bouillaud, according to whom we are left-handed as to language and (perhaps) also to other acts. This view is open to misunderstanding. Bouillaud, however intended no more than to state that we use in speech the left cerebral hemisphere in the same way as a left-handed individual uses his left hand for certain performances, particularly the so-called skilled movements. Obviously, it was a mere analogy: Bouillaud was still far from assuming a neurophysiological basis of right-handedness associated with left-brainedness.[1] Not until 1874 did Hughlings

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[1] A French country physician, Marc Dax and his son G. Dax, must be credited with the discovery (1836) that invariably aphasia is related to lesions of the left hemisphere. This statement, which proved to be one of the most elementary facts in the whole doctrine of aphasia, was the result of the doctor's careful, unprejudiced, and unaided observations made in the rural district where he practiced medicine.

Jackson use the term and concept of the major or "leading hemisphere." In 1905, Liepman identified the corpus callosum as the anatomical equipment for the transmission of excitations from the major to the minor hemisphere. He turned to this transmitting mechanism in order to explain the observation made by him that an individual whose right side is paralyzed is unable to perform purposeful movements with his left hand; this observation with which neurologists are well familiar, has become known as sympathetic dyspraxia of the left hand. Let us retain from these observations that the doctrine of lateral cerebral dominance (of a major and a minor hemisphere) originated from neuropathological evidences; but this proved to be no more than the loss of certain skills after unilateral cerebral lesions. The evidence did not show that these skills themselves must be attributed to the sole activity of the opposite hemisphere. So far, the dominance of one hemisphere over the other, was a mere assumption; a rapid passing from pathological data to conditions prevailing in living and normal man.

In an historical perspective it is not Broca but Bouillaud, who emerges as the inaugurator of the so-called speech center and the rigid cerebral localization which it entailed. In turning now to Bouillaud, I reverse the chronology of our subject. But I am less concerned with the history of events than with the history of ideas. Bouillaud was a follower of Gall and his doctrine of the plurality of cerebral organs. This doctrine is unquestionably the matrix for the later, and still widely accepted, idea of a cerebral activity composed of isolated and elementary functions related to isolated and elementary structures. The neuroanatomical and neurophysiological data which accumulated in the nineteenth and twentieth centuries have furnished such an arsenal of weapons for this idea, that the opposite view--of a total and unitary function of man and his brain--has been condemned to go underground. It remains true, however, that this underground movement does not lack ammunition; such as the indistinctness of the borders of the cortical areas, observed by the eminent Viennese neuroanatomist, v. Economo; or the greater importance placed by the American neurophysiologist, Lashley, on the amount of tissue destroyed compared with the region involved. The main argument, however, rests on thought and critical reasoning. This leads me back to Bouillaud.

Bouillaud's attitude was rather dogmatic; it was determined by several considerations. He believed that the site of the lesion associated with a disordered function is also the site of the undisturbed function. He believed the cerebral convolutions to be the first movers--the true incentives, or the soul, of the vital instrumentalities of articulated sounds. (He thus revived, though unwittingly, the old doctrine of the seat of the soul, which the medical historian is tempted to consider to be the concealed springboard for the whole doctrine of cerebral localization.) Furthermore, Bouillaud felt justified in claiming a speech center, because speech (he said) may be lost without simultaneous paralysis of other motor functions and, conversely, speech may be preserved though there may be simultaneous paralysis of the extremities. He finally concluded that speech must remain undisturbed if parts of the brain, other than frontal, are involved. None of his considerations proved to be indisputable.

The first consideration contains an error of thought. Indeed, we are not justified in deriving the seat of a function from the seat of a lesion. Even a machine can stop running when only the smallest part is damaged. But we cannot conclude from this that the running of the machine or its function has its seat only in the damaged part. A better example is found in a symphony which can lose its desired total effect and its completed structure, when only a single instrument is missing. And again, we cannot conclude from this that the symphony has its seat in the missing instrument. Human language is such a symphony or, to quote von Monakow, a whole of "kinetic melodies." Speech is built up genetically and biographically, and it is the result of an activity spanning broad periods of time. Every cerebral structure shares in this activity. This is the meaning of chronogenetic localization. But this localization does not exclude the fact that language may be disturbed by lesions in certain definite areas. In other words, language is vulnerable to specifically located lesions. Achilles was mortally vulnerable in the heel and Siegfried in the shoulder, but their lives did not reside in the heel nor in the shoulder. It has become increasingly clear that the genetic and biographic structure of language, and above all, the meaning it carries, make it impossible to assign to it a seat--such as a nerve ending or a center of coordination.

In brief, that language can be disturbed or abolished after a regional brain lesion emerges as a fact from a century of investigations. That undisturbed language has its seat in the same region still remains an hypothesis; even an absurd one, since language can have no seat anywhere in space. At best, one may ascribe to the so-called centers the meaning and function of instrumentalities or organs. Such was indeed the opinion of the much slandered Gall, who may be called the illegitimate father of the doctrine of cerebral localization, which with him ultimately rested on no more than the palpating finger. Nevertheless, Gall called his doctrine an organology.

The nineteenth, and above all, the twentieth centuries lost sight of the instrumental nature of an organ, which in contrast to a mere part, is thought to be in indissoluble union with the vital principle. As far as I know, Charcot was the last to use the word organ in the interpretation of aphasia. He believed that "the cerebral hemispheres consist of a number of special organs, each of them having its special function though having intimate relations to the others." Unfortunately, he considered these organs to be no more than separate deposits of auditory, visual, and kinetic images; these different memories were believed by him to have their seats in special areas of the brain. Charcot was still far from thinking that these memory images, or engrams, must be conceived of as carrying in a living organism the criteria of life; subjected to metamorphosis, extinction, maturation, and enrichment--all this as living parts of a living whole with which they are in reciprocal union. This thought is not to be found in Charcot's writings; it appears in those of von Monakow and his school of thought. Aphasia thus understood is not a loss of words, but of the power to use them at will; and the brain is neither a generator of, nor storage house for, words--but an instrument in their evocation.

I stressed the purely instrumental significance of the so-called centers. But an instrument does nothing by itself; it calls for a player. In the same way, an organ calls for man who uses it--and who, in his turn, cannot be conceived of as residing in a minute part of himself. With these statements we have reached the Aristotelian doctrine of life and soul.

Nevertheless, one would do injustice to Bouillaud by accusing him of a naive concept of cerebral localization. In a discussion of our subject, which took place in the late forties in the Académie de Médecine, he warned not to compare the brain with a piano in which each string renders its special sound. At exactly the same time, Brown-Séquard, too, rejected the piano-theory of the nervous system. It would be better, Bouillaud said, to compare the nervous system with the larynx, whose numerous and manifold parts always act simultaneously in order to produce the most various sounds. Bouillaud held this view about 20 years after his first communication on the seat of articulated speech. Since this second thesis is no longer compatible with an authentic interpretation of Gall's views on cerebral localization, one may assume a metamorphosis and, perhaps, a maturation and increasingly critical structure of Bouillaud's thought.

It is true that the results of electric cortical stimulation--obtained by O. Foerster and, recently, by W. Penfield and his associates--seem to speak in favor of a localization of cerebral function. But it was never possible to produce true speech by electric stimulation. The effect resulting from the stimulation of either dominant or non-dominant motor areas consisted of no more than "a sustained or interrupted vowel cry" with an occasional "consonant component," but never an intelligible word. Other stimulation effects were hesitation and slurring of speech, distortion and repetition of words and syllables; confusion of numbers while counting; inability to name with retained ability to speak; use of words somewhat closely related in sounds, or synonyms, or entirely unrelated words. When the patient was talking, the stimulation effect consisted of a total arrest of speech. These aphasic responses were elicited from frontal, temporal, and parietal regions of the major hemisphere. Electric interference involving the right cerebral hemisphere produced arrest of speech, hesitation, slurring, distortion, and repetition.

These observations suggest paralyzing rather than stimulating effects. If this were correct, the electrical cortical stimulation could not be used for illustrating the natural activity of a stimulated area--at least as far as speech is concerned. The result of electric stimulation of the cortex could only be called forth to prove a localization of disturbed or inhibited cerebral function, rather than a localization of undisturbed or pure cerebral function.

Bouillaud's second thesis, that speech remains intact if parts of the brain other than frontal are involved, did not stand the test of time. True, the knowledge of the so-called remote effects was gained many years after Bouillaud. Brown-Séquard was the first to call attention to actions at a distance, and thereby to endanger the doctrine of cerebral localization. It was von Monakow who described them later under the name of diaschisis and who stressed their importance for a better understanding of aphasia.

The principle of diaschisis or cerebral shock has great practical implications. By giving due consideration to the fact that functional disorders may arise as remote effects--that is, from areas not involved directly in lesions, but only secondarily as reactions at a distance--errors in localization and regional diagnosis may be prevented. Speech defects of right-handed individuals may result from lesions of the right hemisphere, throwing out of function the left hemisphere as the result of cerebral shock or diaschisis; but, as a rule, these effects of cerebral shock are transient. Thus the question of cerebral localization has to be answered in a different way, according to the two major stages of brain injuries and brain diseases; those of initial and transient and those of residual and lasting symptoms.

Diaschisis is a transient state of diminished or abolished function, to which regions distant from the region primarily involved may be submitted. Thus, diaschisis is a remote effect, due to the interruption of pathways relating the distant parts to the part involved in a lesion. Diaschisis has not been given the attention it deserves. There are indeed scattered references to this principle in literature, but it has hardly ever been used systematically for a better understanding and interpretation of brain lesions. The majority of publications devoted to the study of cerebral localization still neglect the fundamental distinction between temporary and residual symptoms.

The principle of diaschisis is not the result of pure speculation but the simple and logical expression of what is to be expected after injury to such a highly organized and intricate mechanism as the mammalian brain namely, transitory loss of functions in remote regions What remains as residual symptoms, after diaschisis has passed off, is a combination of the functional deficiencies produced by the destruction and of the functions proper to the spared part--now deprived of the contribution the destroyed part previously made to the integral function It is important to stress that the passing off of diaschisis is a spontaneous phenomenon, involving no training; it thus becomes the true expression of natural recovery, without intentional effort The possibility of a reappearance of neural shock has also been stressed by von Monakow who believed visual agnosia to be a temporary symptom passing to a latent stage under favorable conditions Thus, the doctrine of diaschisis is linked with the doctrine of disease and recovery

In one of Monakow's most famous cases, speech was preserved in spite of the involvement of Broca's area I was given the opportunity to reexamine the brain of this case, fifty years later, and I was able to demonstrate that the regional factor alone is not sufficient to produce aphasia; a chronological factor the so-called momentum or the speed of onset of a lesion, emerged as a concurrent factor of an importance at least equal to the site of the lesion Acute and sudden lesions--such as brain injuries, hemorrhages, or even rapidly growing brain tumors--determine speech defects which do not appear at all or which appear very late in slowly developing lesions; though the so-called speech areas are involved in both instances

In the history of aphasia, the view held by Pierre Marie was of particular importance from the beginning--mainly because Broca's localization of aphasia was criticized systematically for the first time, at least on the European continent. I am not concerned here with the individual steps of Pierre Marie's criticism, which have now become common knowledge I would rather try to uncover the principles adopted by Pierre Marie which, so far, have not been analyzed. Pierre Marie's presentation is distinguished indeed by an exemplary method. He strives for a solution which is intended to be unprejudiced and deprived of every hypothetical and speculative element He is a thorough empiricist and his field is the investigation of so-called pure data

Broca deposited the two brains as confirmatory evidence of his views in the Musée Dupuytren Forty-five years later, Pierre Marie reexamined them and reached the conclusion that neither of them had been investigated thoroughly by Broca either clinically or anatomically In the first of the two cases, Leborgne Pierre Marie discovered, in addition to the frontal lesion a lesion of Wernicke's area and of the lentiform nucleus In the second case, Lelong, he showed that the only lesion was a circumscribed senile atrophy with accumulation of fluid and that, at any rate, during life the patient had shown only senile dementia and not aphasia.

It is to Pierre Marie's empirical thought, and to his rejection of everything except pure data, that we owe his denial of the existence of images and their regional cerebral sites. Indeed, he was one of the first to express this denial in the history of aphasia. Above all, he was one of the first to call attention to certain disturbing observations which cannot help but disappoint adherents of a dogmatically conceived cerebral localization. These observations were later collected by von Monakow and listed in his monumental monograph on cerebral localization as "negative cases"; these were cases of aphasia not associated with a corresponding cerebral lesion or, conversely were regional cerebral lesions not associated with aphasia.

In spite of his empirical thought, Pierre Marie could not avoid transgressing pure data. In fact, the combined anatomico-clinical method, which he adopted, is a method which leaves behind the mere recording of data and which uses the data in order to bring them into a coherent whole. The investigation of pure data teaches us no more than two separate series of observations; the series of the symptoms and that of the lesions. But the correlation of these two series of observations, which is the basis for cerebral localization, is a relationship which is established between the two series, and every relationship is an act of human understanding and human intelligence, but not of mere perception. If correlation were the product of intuition or perception, there would be no argument. Any error in that case would result only from incomplete visual perception, but not from unorganized experience. The century old argument bearing on cerebral localization belongs to the jurisdiction of the understanding, but does not belong to the jurisdiction of the intuition or the so-called pure observation. Whether we want to, or not, we find ourselves here in the area of the first principles of medicine, but no longer in the area of what is material in medicine. Putting aside these presuppositions of the combined clinico-anatomical methods (which hardly could be considered as being still purely empirical), we encounter other tendencies in Pierre Marie which transcend statements of pure data. I am thinking about his extraordinary unitary tendencies which were repeatedly and most strongly reaffirmed by him, and expressed by him in the most demonstrative way when he said: "Aphasia is one," and "Localization of aphasia must also be one."

In his day, only two basic types of aphasia were distinguished: Broca's aphasia and Wernicke's aphasia. The only difference between them, according to Pierre Marie, lies in the fact that in Broca's aphasia the patient cannot speak whereas in Wernicke's aphasia the patient still speaks, though with many errors. But, according to Pierre Marie, patients of both these types are unable to read, or write, or understand difficult questions. This rather cursory conclusion did not survive later criticism. But what I want to stress here is Pierre Marie's strong unitary tendency which ultimately incited him to define aphasia as an intellectual disorder, which is not necessarily limited to speech and which spares emotions and gestures.

Before Pierre Marie, Trousseau had expressed himself in similar though not identical terms. At this point we should remember that Trousseau was also the first who stressed, besides the regional factor, the importance of the nature of the lesion for the genesis of aphasia. He found lasting aphasia to be more frequent in encephalomalacia than in hemorrhages. He anticipated the fundamental difference which von Monakow later established between transient and residual effects of brain injuries. But let us go back to Pierre Marie, who did not accept Broca's thesis that the third left frontal convolution was the site of aphasia. Broca's aphasia, he concluded, is a combination of Wernicke's aphasia resulting from temporal lesions and anarthria resulting from lesions of the lentiform nucleus. The historian of ideas and principles is less interested in this anatomical exegesis than in Marie's dictum, that the degree of aphasia is not in proportion to the involvement of allegedly isolated centers for allegedly isolated language functions, but to the extent of the lesion. He thereby anticipated results obtained later by Lashley; he could as well have referred to his countryman Flourens. At any rate, he was reaffirming his unitary tendency and a shift from a regional factor to a global view of cerebral function and language. The essence of the unitary tendency or global view, that every informed student of aphasia today has in mind, is not reached until language is conceived as rooted in the whole person, his development, his history, his social and educational standing. In my experience, these roots are still demonstrable in the aphasic, who does not cease to be a person, who still has a history behind him, and a future before him--shrunken as his total existence in his present and his future may appear to be. Under favorable conditions, the aphasic might even reach new ways of expression; a field hardly explored so far.

In the second half of the nineteenth century, Hughlings Jackson elevated aphasia to a higher level than mere coordination, once advocated by Bouillaud. However, Jackson did not attract much attention and understanding. Sigmund Freud was one of the very few on the continent who read him, though he did not seem to understand him integrally. At any rate, he missed the Jacksonian purely functional concept of evolution, which Freud conceived in entirely Darwinian terms.

It is one of the most remarkable and distinctive characteristics of Freud's treatise on aphasia (1891) that it was written by an author, who--as he confessed himself in the very beginning--had no personal observation to offer. This makes the book a rare and brilliant piece of medical thought. Though almost entirely escaping the attention of its contemporaries, it aroused the admiration of more than one informed reader of the next generation. The book carries a vigorous polemic element, directed against the then very powerful doctrine of speech centers and one of its most representative apologists, Wernicke.

It is significant that Wernicke's book on the symptom-complex of aphasia (1874) bears the subtitle. "A psychological study made on anatomical grounds." In the concluding chapter of his study, the author reminded his readers of the significant criterion of his theory of aphasia which, in his own terms, rests on the translation of anatomical data into psychological ones. Thus was brought home to the reader, from the very onset, that most unfortunate blending of the two basically different methods of thought which Descartes once strove so hard to distinguish and to isolate. No less significant were the reasons which prompted the author to search for a second speech center, once the existence of the first center discovered by Broca seemed inescapable. Spontaneous or voluntary movements, Wernicke argued, do not instantaneously succeed stimulations; rather they originate from memory images, revived by external stimulations. In speech we have to deal with sound images, the sites of which were shifted by Wernicke to the posterior part of the cerebral hemisphere, believed to be sensory in nature. He focussed his attention on the first temporal convolution, as the presumable site of the second speech center. He felt assisted in this assumption by the results of post-mortem examinations; only as the last link in this chain of reasonings. After having drawn an anatomical blue-print of aphasia, Wernicke tried to deduce from his diagrammatic scheme the various types of speech defects resulting from brain lesions. This was the point of departure for Freud's critical study.

Freud selected Wernicke's conduction aphasia as a test object. This variety was established by Wernicke himself, who attributed its genesis to an interruption of the hypothetical tract connecting the hypothetical auditory speech area with the no less hypothetical motor speech center, the result of this interruption being paraphasia, in the absence of other symptoms. Freud concluded that the disturbance of functions attributed to conduction aphasia cannot be deduced from Wernicke's schema. Thus seemed liquidated Wernicke's hope and intention to analyze and to interpret aphasia in purely anatomical terms; the liquidation was due to reasoning tested by observation.

I am inclined to believe that Jackson was inspired to conceive and to develop his doctrine of aphasia by an observation of stupendous simplicity, namely that an aphasic individual may be unable to repeat after the examiner such simple words as *yes* or *no*, but shortly thereafter, and in the same session, may still be able to use the terms as replies to questions. Similarly, an aphasic may say "Good-bye" when leaving the room, and be unable to repeat the same word after the examiner. Here was demonstrated for the first time in the history of aphasia, *the flexibility of defects.* This meant the end of the famous doctrine of speech centers. It simply is not intelligible that a given speech defect is due to the destruction of a corresponding center, when shortly thereafter the same defect is no longer demonstrable. Here we face on the smallest scale conceivable, the phenomenon of *recovery*. Logically extending the observation, we may conclude that the very fact of recovery speaks against the existence of speech centers. To allow other areas to "take over" the role of the destroyed speech centers would mean to postulate more than one center for the same performance; one may then ask why just two centers? Why not consider the whole brain as one great center? But should the assumption of the vicarious function mean that after the destruction of the so-called speech centers the function would be accomplished by an as yet untrained area, the assumption would equally be detrimental to the hypothesis of specific centers for speech. Besides its fatal effect on the existence of speech centers, the flexibility of speech displayed by the aphasic individual serves to illustrate the two types of language distinguished by Jackson. There is implied in the patient's inability to repeat after the examiner, the affection of Jackson's *voluntary* speech, and in the preservation of the patient's ability to utter the same word under excitement or emotion, Jackson's *automatic* speech. It is in conformity with his law of evolution of nervous function and its reverse under abnormal conditions (dissolution), that the more voluntary functions suffer first and most severely, whereas the more automatic functions suffer last or not at all. He also considered the automatic language to be the inferior one. True speech, he said, is to propositionize. The unit of speech is a proposition; not simply an unrelated succession of words, but a relation in which the terms are modified by each other. It is propositional or superior speech which the aphasic individual has lost, but not the inferior, emotional, interjectional; in brief automatic speech. One will have to bear in mind, however, that occasionally single words, such as yes or no, may assume the roles and ranks of propositions, if other words in relation are implied. This qualification has to be remembered in our attempts to analyze and to understand the true nature of speech, lost or preserved, in aphasic individuals, who may use recurrent or stock sentences, fragments or single words, as emotional or interjectional utterances--but also as propositions.

Jackson reached another definition of language of still greater philosophical scope, destined to assume an all-important place in the future work of one of his most eminent followers. Referring to the term of "aphasia," given by Trousseau to defects as well as loss of speech, and referring to the preferable term of "affection of speech" (including defects and loss), but finally rejecting both terms as inappropriate and insufficient, Jackson stated: "There is often a loss or defect in symbolizing relations of things in any way."[2] It is in this vein that he laid the background for his definition of written language: "Written words are symbols of symbols. . . ."[3] Jackson probably did not know that Aristotle was his forerunner; as a rule, Jackson was more than generous in acknowledging his indebtedness to other authors, past and present. The second sentence in Aristotle's small treatise "On Interpretation" reads as follows: "Spoken words are the symbols of mental experience and written words are the symbols of spoken words."

Henry Head continued Jackson's line of thought. He went a step further by calling aphasia a disorder in symbolic thought and expression. But Head avoided definition of the term. "Symbolic formulation and expression," he said, "are no more than empirical terms invented to designate those forms of behavior which are disturbed in aphasia."[4] We read in another passage: "I am not attempting to set up a new human 'faculty,' an elementary class of conscious processes, or even a primary and coherent group of psychical aptitudes. I use the term 'symbolic formulation and expression' as a convenient designation for the various actions, which are manifestly disturbed as the result of certain organic lesions."[5] In like manner, the terms used in his classification of aphasia (verbal, syntactical, nominal, and semantic) were employed by him to indicate the different types of language problems resulting from damage to the brain.

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[2] Hughlings Jackson, "On Affections of Speech from Diseases of the Brain," Brain, XXXVIII (July, 1915), 113.

[3] Ibid., p. 123.

[4] Henry Head, Aphasia and Kindred Disorders of Speech, 2 vols. (London: Cambridge University Press, 1926), I, 399.

But in spite of the purely indicative nature of these names, and in spite of Head's reluctance to define a specific set of psychical functions or aptitudes, he could not have chosen the term "symbolic" at random. The reader quickly realizes, indeed, that the term was intended to cover a rather large set of "forms of behavior," such as words, numbers "and other analogous symbols." Head's classification of aphasia is a classification according to language in its own terms, but neither according to neuroanatomical nor to neurophysiological criteria.

None of the subgroups of aphasia distinguished by Henry Head was intended to be understood as a separate entity. Each of them includes more functional disorders than were expressed by their names. In fact, the actual picture of aphasia is determined by individual talent and skill in speaking, reading and writing. I know of no case of aphasia ever observed in an illiterate. Moreover, the picture changes according to the stage of the patient's aphasia, whether early or late. Finally, we must remember that the patient's performance will break down early when confronted with a difficult task in life or a difficult test in examination, no matter what variety of aphasia.

Taking into account all these factors, one understands that Head himself ascribed but little importance to his scheme of classification. Classification is, indeed, no more than an aid, serving preliminary orientation in a still unexplored area. Even in this perspective, classification often obstructs our insight into the dynamics of brain lesions. But it is precisely these dynamics which matter in the reeducation of speech; reeducation cannot be successful unless one knows the laws presiding over the loss of speech in each individual case.

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[5] Ibid., I, 504.

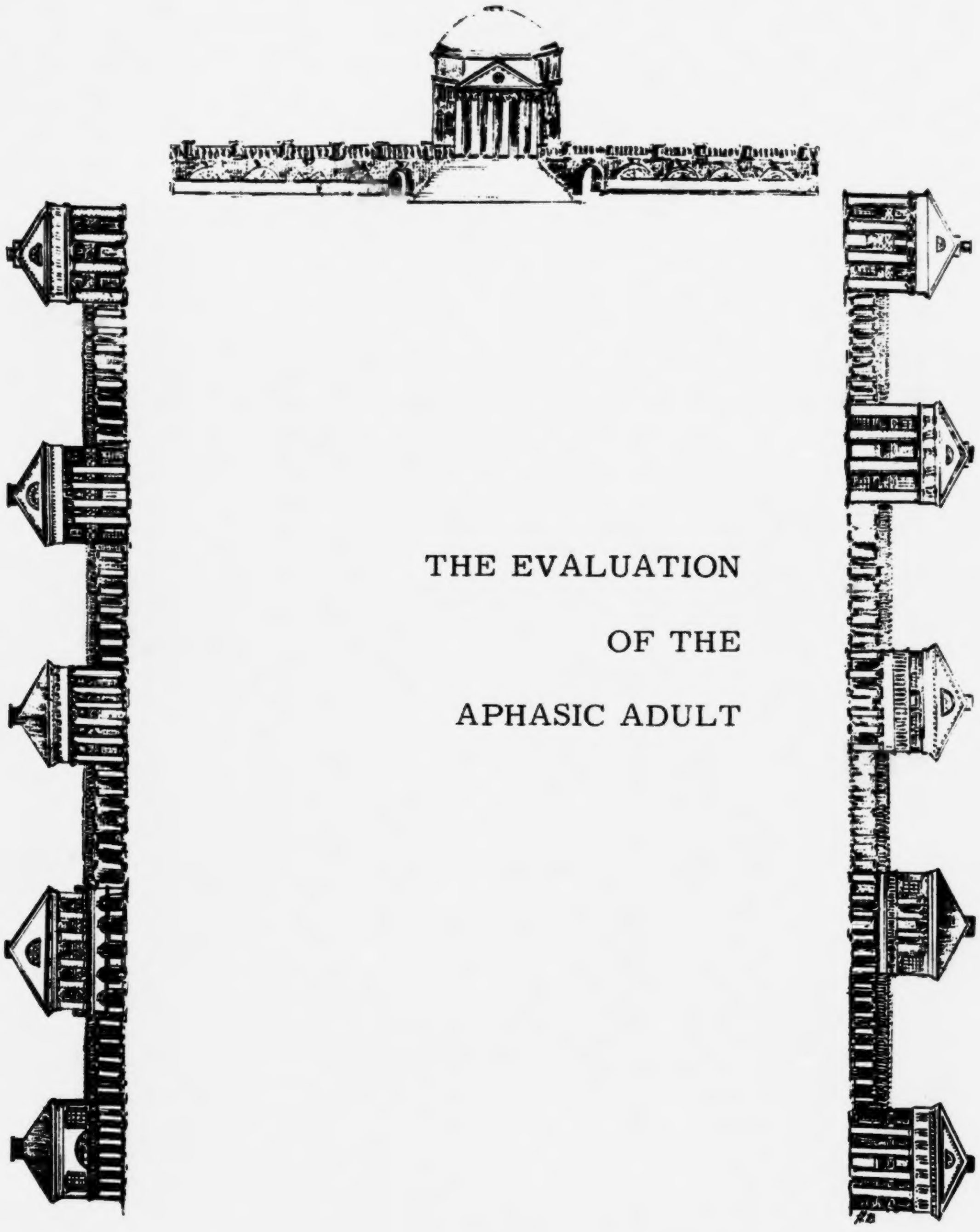
Jackson as well as Henry Head considered cerebral localization of aphasia with caution and reserve, but they rejected outright cerebral localization of speech. It was especially the flexibility of defects, seen in the course of the same examination where the brain lesion remained the same, that determined their rejection of an invariable dependence of speech on circumscribed structures. We encounter the phenomenon of restitution of speech here, to which (strangely enough in the history of aphasia) a much less important role was left than to the defects of speech. At this point the observations of polyglot aphasia claim our attention. The preservation, and regaining of one or more languages, which need not be the mother tongue, can no longer be explained purely anatomically. They require, in order to be understood, biography and situation.

We have to concede that few authors have accepted the view that speech can have no regional localization in the brain. Even Pierre Marie, who rejected congenital or preformed speech centers, at a later date was inclined to accept at least adaptation centers--which he conceived as being individually acquired by training, on the model of athletic or musical performance. This would be a Lamarckian view, the correctness and scope of which it is not my task to discuss at this time. Pierre Marie conceived such an adaptation center of speech as being a cerebral structure of association. But it is doubtful, to say the least, whether one can conceive of language as being composed of associations; of connections of finished elements, such as images and memory images. Such a sensualistic view would ignore the creative element which is implied in human language, and which is expressed in the meaning of a word. This meaning is more than a connection of preexisting elements. For these and other reasons it is impossible to explain aphasia satisfactorily by descriptions of associations.

Turning finally to the recent history of aphasia and living pioneers, time allows me no more than a mention of Kurt Goldstein. He is one of the foremost representatives of the view that neither the structuring of language nor the breaking down of language can be explained by elementary functions--such as visual, acoustic, and kinetic images; their hypothetical anatomical structures and their equally hypothetical associations. He referred to von Humboldt's concept of language as the criterion and manifestation of human nature. As were some of his predecessors, Goldstein is convinced that aphasia is the result of a fundamental and most general disorder. But unlike Trousseau and Pierre Marie, who were satisfied with the assumption of a rather vague intellectual disorder, Goldstein strives for a more precise definition of this disorder. He calls the aphasic an individual who is sinking from a more abstract towards a more concrete behavior, and whose language disorder reveals defects of function also displayed in other fields of performance. Symptoms which we observe in circumscribed fields of examination are always partial manifestations of a total disorder. All failures are interpreted, by Goldstein, as an impairment of abstract attitude. But since a given task may represent a concrete situation to one patient, and an abstract situation to another patient, room is left for an individualizing evaluation of brain damage, and its effect on speech.

It is the same basic disorder, we learn, that is at the root of aphasia, apraxia, agnosia, disorientation, inattention, even dementia and emotional dullness--according to the performance field tested. Thus it seems that the independent separate existence of many a brain pathological syndrome is threatened.

Indeed, we have reached the limits of a synthetic view of nature. It would be illogical and unfair to expect, from such a synthetic view, information which can only be obtained from analytic methods. We here face a thesis and antithesis discussed at a memorable meeting of the Paris Royal Academy of Science, on February 22, 1830. Thesis and antithesis were subjects of heated polemics between Cuvier and Geoffroy de Saint-Hilaire. Goethe devoted to this polemic his last work which bears the title, Principles of Zoologic Philosophy, and which bears the date, March, 1832. At the very beginning of Goethe's article we read: "The permanent conflict between the two modes of thinking which has divided the world of science for so long is found here"; by which he meant, here in this conflict between the generalizing tendency of Geoffroy de Saint-Hilaire and the particularizing tendency of Cuvier. Goethe believed that the problem, and its discussion in the French Academy, was of greater importance for the history of human thought and culture than the political events which disquieted the population of Paris at that time.



THE EVALUATION
OF THE
APHASIC ADULT

78-80

NEUROLOGICAL EXAMINATION

Fritz Dreifuss

It is my task this morning to conduct a neurological examination on a patient who was entered with the complaint of aphasia I might just preface the examination with a few remarks bearing on yesterday's discussion You will remember that in the afternoon Dr Walther Reise gave us a little cautionary tale, the tale of Paul Broca who made a good observation and then was led over the course of a couple of months into making untenable inferences from the observation--something which we are sometimes liable to do and of which there has been so much in the realm of aphasiology. As clinicians we make many observations, some of which do not carry with them, at that particular time, any ready explanation and which we have to store away until we have collected more and more and are able to see a developing pattern. It is only by careful observation that we can supply the perspective and the delicate brush work which are necessary for painting the complete picture. At the time we apply the brushstrokes (the patient in his careful introspective account of his illness, as Dr. Eisenson has pointed out, provides the isolated brushstrokes which at the time may be disconnected) they are as it were atomic and it is only later that we see the whole picture evolving, that we see why things happened as they did. The importance of the patient's introspective account is paramount. He is the only person who can communicate to us what were his particular impressions at the time--his sensations--and whether language was involved alone or whether thought was disrupted as was language.

In the neurological examinations, we are concerned with answering certain problems. The first question we ask ourselves is Where is the lesion which is causing the patient's disability? We heard yesterday that there are certain functions which we can definitely localize in the brain, and there are other functions--such as speech--in which definite cerebral localization is tenuous. I think it is better to think in terms of cerebral specialization than in terms of cerebral localization; we can say that there are certain parts of the brain that are specialized towards a certain function, rather than that a certain function is localized in such or such a gyrus. There is, however, in the brain such a thing as point to point localization. For example, if we look at an object then every portion of our retina is represented pointwise in the occipital cortex, and if we stimulate by pain or touch the finger tip or any other part of the body, that part of the body has a point to point localized area in the thalamus and the receptive sensory cortex. When these impulses reach the cortex, then we get integration of these percepts into concepts and it is at this stage that the point to point localization concept becomes inadequate. In the case of speech, likewise, the auditory apparatus is very much involved with the receptor organ in the ear, the cochlea, and the auditory pathways to the cerebral cortex where again there is point to point localization in the superior temporal gyrus of various areas of the cochlea, so that the apical parts of the cochlea which receive the base tones are represented more anteriorly and the basal parts of the cochlea which receive higher tones are situated more medially and posteriorly. It is only after this that integration takes place and we begin to form concepts from these auditory percepts, and here we are not at all sure of our localization except we know that the area in which this occurs is somewhere in relation to the superior temporal convolution in the auditory cortex. These concepts that are formed are, of course, individual to each one of us. If I speak of a certain town, for example, this does not conjure up in every person's mind the same picture. Some see in their mind's eye: people, streets, and shops. This town to other people is just a dot on a map or an airport at night, so that the concept of this particular message is a different one in each one of us. Therefore, what we say and what is conceived by the recipient of our message may be two somewhat different things.

To go back to our neurological examination and the answer to our first question, the patient comes to us with a certain history of disease and we try and localize so far as possible, in this patient's nervous system, the situation of the lesion which has given rise to his complaint. This is the primary object of the neurological examination. The second question is: What is the disturbance of function produced by this lesion? In other words, what is the physiologic disruption which this disease process is producing in this particular patient's nervous system? And thirdly: What is the lesion? What is causing this patient's disease? Now in examining one of our patients we draw certain inferences from observations which I think are valid, because they have stood the test of time. They have been correlated, pathologically and anatomically. We are not like surgeons, who can lay bare the part that is diseased to look at it. We have to look at a patient who is intact; whose nervous system is enclosed in a bony box, and we have to look at the disease's manifestations as produced peripherally without being able actually to look at the lesion. We have to observe the disturbances of function. In this, aphasia like epilepsy is but a symptom; a fragment of the larger disturbance which we must fit into an over-all neurological picture. Only after we have grasped the over-all neurological picture can we allow ourselves the luxury of going back and playing with each particular symptom and try to explain it. What we see in the neurological examination is the reflection of certain parts of the nervous system, according to the function of that part of the nervous system.

Perhaps more importantly, we also must find out what is the nature of the disease which is causing this disturbance; and here we are entirely dependent on the patient's history. The taking of an accurate history is probably the most important single step in the neurological examination, if one were deprived of everything else one could still make a fairly accurate diagnosis by taking enough time talking to the patient and getting an accurate history--the patient's accurate account of the way in which things happened to him.

The patient today is a 52 year old right-handed man; an administrator in a large manufacturing plant; an educated and intelligent gentleman. He was in his usual good health until, one day in May of this year, he suffered a coronary thrombosis. After three weeks in the hospital, he was discharged home; and perhaps rather early following one of these things, returned to work. Within two weeks of returning home, he developed severe epigastric abdominal pains and entered the hospital in his home town in a state of shock, and with an aneurysm of his heart; a bulge in the weakened heart wall. While in the hospital, he suffered the attack which brings him to our notice today. He suddenly experienced the inability to use his right arm and leg, and the inability to express himself in speech. Over the next few days he was in this state of a right hemiplegia and aphasia, and a few days after this he developed a further episode consisting of sudden pain, numbness, and coldness of his right leg. What had happened was that from the aneurysm in his heart a little clot became detached and found its way into his left middle cerebral artery, producing his right hemiplegia and aphasia. A few days later, a further such clot entered the femoral artery supplying the leg. He suffered what we discussed yesterday as an ischemic lesion; one that is brought about by the lack of blood supply to a part of the brain. Later, the same thing occurred to the right leg. He was admitted to the hospital where he was examined and started on anti-coagulant therapy. At that time, he was found to have a severe right hemiplegia, hemihypesthesia, and hemianopia. His speech problem was a moderately severe expressive dysphasia with a receptive dysphasic element in that, although he was able to understand most of what was said to him, he had little awareness of his own speech difficulty. This resulted in a jargon aphasia.

> (At this stage, the patient was introduced and a complete neurological examination was demonstrated, which showed that there was a minimal residual right hemiparesis, a mild right hemihypesthesia affecting predominantly cortical sensory modalities, and a mild intellectual deficit as evidenced by the inability to repeat the Babcock Sentence or to retain more than four digits.
>
> The patient was questioned in detail about the onset of his dysphasia and it was elicited that he had been unaware of the difficulty, even when it had been pointed out to him that he was unintelligible. He first realized his deficit during detailed aphasia testing. Thereafter,

he was aware that his thoughts outpaced his ability to express them. He is now quite aware of his residual difficulties, including his intellectual blunting, in that he is now able to perform only relatively menial tasks instead of his previously responsible occupation.)

In summary, we have elicited from the history, the nature of the disease process from which the patient suffered. On the basis of the clinical neurological examination, we can be reasonably certain of the area of the brain which was affected by the lesion. One can further infer which cerebral blood vessel was occluded by the embolus, and we have specialized radiological tests available to demonstrate this visually by means of arteriography.

LANGUAGE EXAMINATION

Jon Eisenson

(After administration of Examining for Aphasia[1] to patient)

Well, then, let me go over the results with you and give you a little bit of the philosophy behind this particular examination instrument. I think all of you appreciate that an instrument represents a philosophy of the person who has devised it, and that there are different philosophies. The philosophy of Joseph Wepman's Modality Test is based upon his concept of the nature of aphasia. Wepman's concept is based on Osgood's model of what language is about. Wepman's Modality Test represents a particular philosophy of aphasia. Hildred Schuell's test has a different philosophy or rationale. My test was devised in order to get some idea of what the patient is able to do--to provide an assessment of his abilities, his liabilities, his strengths, his weaknesses--all with a view towards getting him started in therapy. This inventory was not devised, as was Wepman's, to get kinds of responses that are based upon a philosophy that language consists of modalities; that you can get responses that fall in or out of modality categories. Though our philosophies differ, I think that virtually any of the sub-categories of my test can be translated into one of the modalities of Wepman's test. With my inventory, I begin by trying to arrive at what I call sub-aphasic abilities or disabilities. So we begin by testing for the agnosias.

Agnosia is a disability in recognition through a sensory avenue, either when the sensory avenue as such is intact or where the disability found is over and above any existing impairment of the particular sensory organ. For example, in the case of our patient today, his tactile sensitivity is weakened, but it is not absent. Now suppose he confuses a poker chip with a half-dollar, when these articles are placed in his hands and while his eyes are closed. We could attribute this disability to agnosia. How can we account for our patient's confusion between a quarter and a nickel? He does feel, he does know that it is a coin, but beyond that he does not know what kind of a coin it is. If he were able to match the two, then I would say we could overlook the agnosia as a possible defect. But we must test for agnosia. If a patient has a real agnosia--if he cannot recognize a sensory pattern through a particular avenue--then certainly he cannot evaluate the pattern. Evaluation begins after you get recognition and discrimination. I begin testing for the agnosias, however, on a high level. I do not begin by testing for agnosias on the lowest possible level; too often it is a waste of time. If I permit a patient to tell me that one object is a pencil and another is a knife then I know, by this response, he does not have an agnosia for the modality or avenue of presentation. If he does not respond correctly, or if he fails to respond verbally, then I can always drop to a lower level to evoke a response. The lowest level is usually that of matching like objects. If he can match them through the particular modality which we test (testing must be done through one modality at a time) then we may be confident that the patient has no agnosias through that particular modality. So we test for the visual, the auditory, and the tactile.

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[1] Jon Eisenson, Examining For Aphasia, Rev. Ed. (New York: The Psychological Corporation, 1954).

There is one little test--reduced size pictures--that does not really belong in those for agnosias. I include this test because it provides me with very interesting information. If you recall, when I present this test, I change the rules and instruct the patient: "Don't say anything to me, just point. Show me which pictures are the same. They are not exactly alike but almost the same." Usually, if I have any difficulty in getting responses, it is between these two: scissors and shears. I frequently find that a patient often has to say to me "Well, yes but no"; which is alright. He is telling me that the items are not exactly alike but still not different; they have essential similarities. Some patients, however, reject completely any possibility that these are alike. Some patients have to verbalize how they feel about accepting this test item. If they are able to verbalize, they may say: "Well, this is for cutting paper or cloth and this is for tin"; which is a good response. The significance of the response depends upon what kind of a patient we have.

If the patient is a tinsmith he will reject the likeness, because to him scissors and shears are functionally different. But most patients do accept the likenesses. Some patients have to verbalize and have to become quite specific and answer: "Yeah, they're alike, but this one's darker." The more specific and more detailed the patient has to be, the more he informs me that in order to come to terms with the situation he has to modify the terms presented to him. This is in the direction of what Kurt Goldstein talks about as making a situation concrete, and what I imply about making a situation ego-oriented; and so acceptable to the personality of the patient. I have found that the patients who reject completely almost alike pictures, who just do not see similarity, are also unable to do the pretended action items in the second part of the test. (For example: "Show me how to drink a glass of water"; when the patient has no glass.) Occasionally a patient can do the pretended actions; can give you a meticulous pantomime. For example, when asked: "Show me how to smoke a cigarette," the patient may reach into his pocket, pretend to draw out a cigarette, and go through a very careful pantomime. There again, the more a patient needs to create a reality situation for himself, the further away he is from what Goldstein talks about as having an abstract attitude, and what I talk about as being unable to adopt the cultural assumption of a situation. The patient must make it an ego-oriented situation for it to be acceptable to himself. Such a condition has implications for therapy because this kind of a patient is going to have difficulty with what you mean by language; with what you mean by words. The "word game" to him is different than it is to most of us. Some patients are simply unable to play the "word game" as most of our culture plays it. When we say "What do you wear on your head?" we mean not your head but "What do people wear on their heads?" The word you, in this verbal arrangement, is usually regarded as indefinite, rather than specific. Most of us know what we mean by this arrangement of words. The patients who cannot accept this implication of meaning are telling you a good deal about themselves. To continue from this tangential intrusion, I get very interesting responses sometimes to the whistling item of the agnosia group.

For example, when I whistle and the patient says to me "That's a wolf," I do not mark the response wrong. I think he has a lot of insight or maybe he is keeping his courage up. I do not say he has an auditory agnosia at that particular point. He has let me know that he knows what a particular whistle-sound means to him. The item of "Put your left hand on your left elbow," I throw in also to see whether the patient gets the idea; understands that it really cannot be done. (Incidentally, I do not ever ask for the defective hand, because you may then get a rejection of the task. I ask the patient to use only the hand that is not impaired.) Some patients try quite seriously to do what is asked. Others simply put the left hand on the right elbow as if to assume that is what any sensible person would have asked. Such a patient is giving you the benefit of the doubt. If this happens, I repeat: "Now what I said was put your left hand on your left elbow." And I wait for the reaction with a smile. Your voice must smile, and you must smile, when you test for this "no can do" item. You must look amused at the effort--it helps the patient to get the point. If he fails because he thinks you are serious, to save face he may keep on trying. Perhaps it is your face rather than his own that he is trying to save.

We then go to the testing for auditory aphasia; Auditory Verbal Comprehension. Notice that for the item "In what month is Christmas?" the patient said "December 25th." His consistent tendency was towards the highly specific. In the oral paragraphs our patient called the bird "Jack" instead of "Jackie"; which is alright. He used the word active instead of lively; which again is alright. He got the idea, but I wanted to see whether he could get the synonym for it, and he accepted lively as an appropriate synonym. In the paragraph on maple sugar, I think he got the correct sentence, but you notice he changed the sentence. The answer "It takes a lot of sap to make a little sugar" was presented by him as "It took a lot of maple sugar"; which I would accept as a possible paraphasic error but correct for the item. He understood the question and his answer was correct even though he changed the words. In the next paragraph, the poetic one about the twilight and the mountains, he did not get the word twilight but he gave me the equivalent of late afternoon. He correctly said "mountains" for the item "What could be seen against the glow of the sky?" For "What color did the mountains appear to be?" the correct answer is "white" and he said "brown." We may conclude that he did show some degree of error in the more difficult paragraph. In silent reading, he got all the sentences right. In the paragraphs he got the first paragraph right; the one about Ted who worked hard all morning. The second paragraph was also correct. He made a spontaneous correction on one of the items; which was good. In the last paragraph, a fairly difficult one, he got two out of four answers correct. So he does show some impairment in reading, with the impairment increasing as content increases, both in the sentence and the paragraph material, and whether presented auditorially or visually.

In testing for apraxias, our patient had no difficulties. He even helped me with the padlock. He did the pretending action pretty well. As for the verbal apraxias, he was able to say 1, 569 (one thousand five hundred, sixty-nine) which has more syllables than 3-1-7-5-9. I think the explanation is that he is dealing with ideas in one, and dealing with isolated digits in the other, and he is able to tie more meaningful syllables together than he can a rote reproduction of digits. This is not surprising. If we recall items on tests of intelligence, children can reproduce many more syllables in a meaningful sentence than they can syllable digits that are not meaningful. The patient had difficulty in saying "Methodist Episcopal Church." Before we accept this difficulty as being evidence purely of dysarthria, we should always give the person a chance to say the negative. In this instance, when asked to say the negative you recall that he had less difficulty. He had to tell me he was a member of the Catholic Church, not the Methodist Episcopal, or the Baptist. When I changed the sentence to "I do not belong to the Methodist Episcopal Church," even though we had a longer sentence, he had less difficulty in repeating it. Now, how shall we interpret this? Goldstein, I think, would interpret (or would have interpreted it once) to have said you see this is an abstract question that you ask--The Methodist Episcopal Church--this is an abstract concept; the patient cannot get it. But when you say "I do not belong," this is a reality situation for him and he is able to accept that fact that he does not belong to the church. It violates him to say that he belongs to a church to which he does not belong, but about which he may have real feelings. I am inclined, in this instance, to agree with Goldstein that such a response is an indication of the loss of abstract attitude, or at least a disinclination to the use of the abstract, and an assertion of a highly concrete attitude. My interpretation is a bit different from Goldstein's, but I think the difference is important. I consider the change indicates a loss of ability to adjust to cultural demands and cultural expectations, in terms of language, and a replacement by an ego-orientation in regard to the use of language. If language can be personalized the patient is able to say what he needs and wishes to say. If it is not personalized he has more difficulty in adjusting to linguistic situations. Sometimes he goes along with this person's language; he plays the game very well. But give him a chance and he tends to become personalized in his responses. I think this is something we must recognize in this patient and recognize in his recovery process. This will have implications and limitations for what recovery he can be expected to make, the direction of the recovery, and what language will mean to him--both on the receiving end and on the productive end.

For automatic content he was able to go from one to twenty. He did block on the i in the alphabet. The days of the week were correctly produced when I created a set for him. He was correct on the months of the year. Our patient had considerable difficulty in singing. His hum was a tuneless hum, yet his speech does not lack conventional melody. If his speech lacked conventional melody, I would say that this is possibly part of a dysprosody, a breakdown in ability to deal with melody. But his speech does not lack melody; he was able to get inflections and intonations, and the innuendoes of my speaking, so that it may well be that he is a little bit on the self-conscious side. On the other hand he did not know his hymns. It could be that he was just afraid to reveal a memory defect, and for this reason he did not undertake the tune. I should have hummed the tune and let him repeat the hum. But this is always a test of my own ability, and I must confess to you that since I was in the first grade I was always in the listener section.

He made no errors in writing numbers and his letter writing was accurate, except that he did have difficulty with u. He wrote it twice and corrected it. With spelling, the first word that I said was "tell"; he wrote let. This is an interesting reversal, make what you will of it. I am not sure, because it was the only reversal I noted and there are four words there that permit reversal. They are included for this purpose. The words tip and on and was are there to give the person a chance to reverse. But he did not. I gave him a sentence "I do not--"; he wrote the do not but did not write the rest of the sentence. He wrote on and suppose correctly. For campaign he wrote can be. He first spelled the word foreign as forieng and then changed it to foriegn. I gave him an opportunity to go back and check but he saw nothing wrong. For "This month is December" he wrote September. When I gave him a chance to go back and correct, he seemed to be aware that something was wrong, but he wrote over the letters of September without changing them. He correctly wrote the sentence "Most girls like to sew." "Mexico is south of the United States" was written "Mexico is south of America"; which Mexico might resent. He named the body parts correctly.

When our patient was asked the question "What do you wear on your head?" the answer was not a hat, but my hat. He maintained the personal possessive pronoun; he insisted on the my. Most of you would not personalize it that way. You would accept the question as a general question and give a general answer. But this patient, quite correctly from his point of view, accepted it as a question addressed to him as an individual. The you was not general you, the you was specific you. When a patient gives you a response, you must at any particular moment try to figure out the dynamics behind the response. What are the dynamics of the error? What are the dynamics of a correct but rather unexpected response? Why does the patient say the kind of thing the patient says? Our patient's responses often tend to be along a direction of ego-orientation. To understand him--to understand why he gives you this response instead of another kind of response--you must understand his special orientation. His responses are correct, but unexpected, until you appreciate the dynamics of his orientation.

He made a surprising number of errors in arithmetic: 7 and 3 were 10; 5 and 13 were 18; he divided correctly 21 by 3; and he divided 24 by 4; but 9 by 6 became 114, and for 14 minus 5 he said "one" I guess he must have thought of it as 4 rather than 14. He got the orange problem wrong, the others were correct; even the item of the bananas costing 3 for 10 cents. He did this rather quickly. This indicates, I think, that when he does things automatically he gets them right, but when he begins to figure them out he makes mistakes.

I think it was interesting that he made errors in the first paragraph of the oral reading, but not in the second. I think possibly in the first paragraph he was anticipating what the words might be and he was doing anticipatory reading. He was reading for meanings he thought should be present rather than for words actually present. He could not anticipate the words of the second paragraph so he stayed with the context.

Well, these are my impressions. What kind of a patient is he in terms of recovery? I think that he will improve considerably. I believe that he will continue to recover spontaneously. He will probably recover a little faster with help. I think that his ultimate recovery will be limited by his tendencies for personalization, by his ego-orientation. He will probably continue to have difficulty with abstract notions. A clinician will need to be careful to discriminate between the abstract and the highly personalized. This patient needs to be approached through his ego; it is a large ego, that has a large body to encompass it, and we must nurture the ego. If we overlook it, we will not get the most out of him. I think, however, that with motivation we can make changes for the better. I think at a particular point in therapy it should be possible to motivate him to drop the my by pointing out that "most people wouldn't bother saying it that way. Most bright people (and I would emphasize the bright, so that he might identify with bright people) would just merely say 'hat', instead of saying 'my hat'." I would give him a chance again to come to terms with his own ego, and to identify with the bright person he probably was and potentially may still be.

PSYCHOLOGICAL EXAMINATION

James I. Lore

A thorough psychological examination evaluating the intellectual and emotional resources of the aphasic patient constitutes a first step in treatment and provides a baseline from which to measure subsequent progress. The psychological tests used in this evaluation may be divided into five categories: (1) intelligence, (2) special disability, (3) personality, (4) educational achievement, and (5) vocational interest and aptitude.

Before the patient is tested, his physician should be consulted. Information concerning the patient's physical limitations should be taken into consideration. Furthermore, there are often times in the patient's routine in which testing might be more or less feasible. And finally, the patient should have eye glasses, hearing aid, and so forth, available when the testing is attempted.

Tests of intelligence----Tests of intelligence are usually administered to establish the patient's intellective capacity and function. In cases of organic brain pathology, these tests are particularly useful in indicating the extent of deterioration or impairment and in revealing the emotional reaction of the patient to this change in intellective function. Either the Binet[1] or Wechsler[2] tests are included in most psychological batteries and would be useful with most mildly involved patients. When the communication problem is so serious that the patient must indicate his responses by pointing, the Progressive Matrices[3] and a picture vocabulary test, such as the

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[1] Lewis M. Terman and Maud A. Merrill, Stanford-Binet Intelligence Scale, Form L-M (Boston: Houghton-Mifflin Company, 1960).

[2] David Wechsler, Wechsler Adult Intelligence Scale (New York: The Psychological Corporation, 1955), or David Wechsler, Wechsler Intelligence Scale for Children (New York: The Psychological Corporation, 1949).

[3] J. C. Raven, Standard Progressive Matrices (London: H. K. Lewis & Company, Ltd., 1938), or J. C. Raven, Coloured Progressive Matrices (London: H. K. Lewis & Company, Ltd., 1947).

Full Range Picture Vocabulary Test,[4] the Peabody Picture Vocabulary Test,[5] or the Quick Test[6] may be used. By comparing the patient's recognition vocabulary with his ability to analyze and synthesize (as measured by the Progressive Matrices), it is possible to gain some indication of the patient's residual assets and his prognosis in rehabilitation. Certainly, it has been this examiner's experience that the patient who achieves a more substantial level on the Progressive Matrices than on the picture vocabulary test has the best prognosis for rehabilitation.

The Binet or Wechsler test will, of course, provide much more interesting and varied data on the patients who are capable of completing them. For example, several of the subtests of the Wechsler are particularly sensitive to organic brain pathology, and the patient with even mild pathology may clearly show a pattern of response peculiar to patients who have organic problems. Furthermore, the inter-test scatter, intra-test scatter, and the quality of language the patient uses in responding may also be consistently peculiar to patients with organic brain pathology. Finally, of course, the nature of specific responses may reflect organic brain pathology. So tests of intelligence may indicate not only the extent of intellectual deterioration or impairment but also some specific facts which may be useful in determining the etiology and prognosis of the organic brain pathology.

In those cases in which it is impossible to obtain a valid evaluation from the patient's performance, it may be advisable to use the Vineland Social Maturity Scale[7] to evaluate the patient's level of function. This test is not administered to the patient; instead, the examiner interviews persons familiar with the patient's behavior and records the results of his interview. He then evaluates the information he has obtained in terms of the numerous behavioral tasks such as: "Asks to go to toilet" and "Writes occasional short letters." The result is a "Social Quotient" similar to the Intelligence Quotient of the Binet type test. The test is often very useful with severely handicapped individuals and it provides information and data which are carefully arranged in developmental sequence. Merlin J. Mecham has recently prepared a test[8] using only the communication items from the Vineland, which may be useful when only these items are of interest to the examiner.

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[4] R. B. Ammons and H. S. Ammons, Full Range Picture Vocabulary Test (Missoula, Montana: Psychological Test Specialists, 1948).

[5] Lloyd M. Dunn, Peabody Picture Vocabulary Test (Minneapolis: American Guidance Service, Inc., 1959).

[6] R. B. Ammons and C. H. Ammons, The Quick Test (Missoula, Montana: Psychological Test Specialists, 1962).

[7] Edgar A. Doll, Vineland Social Maturity Scale (Minneapolis: Educational Test Bureau, 1947).

Tests of special disability----Tests of special disability are usually administered to determine the ability of a person to demonstrate his adequacy in some specific function. The Wechsler Memory Scale[9] is a good example of this type of test. It taps various aspects of memory functioning. It is a necessary supplement to the Wechsler intelligence tests and is often given in conjunction with them.

The Illinois Test of Psycholinguistic Abilities[10] is basically used to provide a diagnostic measure of language development in children from the ages of 24 months to 71 months. While this test is still in experimental form, it affords many items which can indicate the nature and extent of the patient's communication problem and thus give the therapist a more specific notion of the instructional level and the frustration level of his patient's function. Furthermore, as with the Progressive Matrices and the picture vocabulary tests, many of the items involve only pointing to the selected response and thus eliminate some of the effects of aphasoid pathology.

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[8] Merlin J. Mecham, Verbal Language Development Scale (Minneapolis: American Guidance Service, Inc., 1958).

[9] David Wechsler, Wechsler Memory Scale, Form I (New York: The Psychological Corporation, 1945).

[10] S. A. Kirk and James J. McCarthy, The Illinois Test of Psycholinguistic Abilities (University of Illinois: Institute for Research on Exceptional Children, 1961).

The Color Form Sorting Test[11] gives information as to the patient's ability to make simple perceptual shifts and to verbalize his thinking. It is a valuable addition to a complete examination of intellectual functioning. The patient is presented with 12 tiles, four of which are circular, four triangular, and four square; one of each shape is red, one green, one blue, and one yellow. The patient is presented the tiles in random order and is asked to arrange them so that the ones that are alike are grouped together. If he comprehends the task, he should then sort the tiles according to color or form. Usually, the more regressed patients will choose color first. After his initial sorting, each patient is asked to explain the reason for his sort. The tiles are again presented in random order and he is asked to group them in another way in which they are the same. He should respond by sorting according to form or color, whichever differs from his original sort, and should again explain the reason for his sort. Color sorting can be done by a patient with a mental age of four or five years, form sorting requires a somewhat higher mental age. The normal subject should be able to make the perceptual shift at a mental age of about ten years. The patient with organic brain pathology is usually able to sort either according to color or to form, but is often unable to shift to the opposite concept. If this test is repeated as the patient improves, evidence of his progress may be clearly apparent in his reacquired ability to make perceptual shifts.

The Bender Visual Motor Gestalt Test[12] consists of nine geometric forms, each drawn in black on a small white card. The patient is asked to copy each of these forms as well as possible on a sheet of unlined paper, using a pencil with medium lead and a good eraser. Of course, many subjects are reluctant to draw anything, and some indicate that they cannot do so because of such factors as tremor, hemiparesis, or lack of skill. However, even if the patient has great difficulty in reproducing the gestalt, he should be encouraged to do so. The test is often useful as a screening device to indicate the presence of severe levels of regression, organic brain pathology, and social-emotional adjustment problems, consequently, some examiners prefer to initiate a battery of tests with this simple device and to determine the subsequent tests to be used by the patient's performance of these tasks. As in all tests, the patient's reaction to these tasks is of great importance and it is wise to note his comments and nonverbal inter-action with the test materials.

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[11] Kurt Goldstein and Martin Scheerer, "The Weigl-Goldstein-Scheerer Color Form Sorting Test," Psychological Monographs, LIII, No. 2 (1941), 110-130.

[12] Lauretta Bender, Visual Motor Gestalt Test (New York: The American Orthopsychiatric Association, Inc., 1946)

Tests of personality----While there are many tests of personality, the Rorschach,[13] Projective Drawings,[14] and the Thematic Apperception Test[15] are the three most frequently used with aphasic patients. The Rorschach test is a series of ten white cards containing blots of ink. The patient is asked to indicate what the ink blots appear to be and anything he sees in them. Subsequent questions indicate what portions of the ink blots he used in his perceptions, what about the ink blot drew his attention to the perception, and what, if any, unusual aspects of the perception were apparent to him. This test gives a sample of the patient's perceptual functioning. It requires him to impose meaning on relatively ambiguous visual stimuli. The Rorschach is a highly technical and sensitive instrument; it may sometimes reveal residual assets that other tests fail to detect; it may be quite consistent with other tests, and it may amplify the findings of other tests. It is neither simple nor easy to interpret but, when the patient is capable of responding to this type of task, it is most useful in providing data concerning the nature and the extent of the impairment and in revealing pre-morbid and residual personality data.

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[13] Hermann Rorschach, Psychodiagnostics (5th ed.; Berne, Switzerland: Hans Huber, 1942).

[14] For a review of frequently used Projective Drawings techniques, see Emanuel F. Hammer, The Clinical Application of Projective Drawings (Springfield, Illinois: Charles C. Thomas, 1958).

[15] Henry A. Murray, Thematic Apperception Test (Cambridge, Massachusetts: Harvard University Press, 1943).

The Thematic Apperception Test consists of a series of pictures to which the patient is asked to respond with a story, indicating what events led up to the picture, what is happening now, and what will happen in the future. While the fundamental purpose of the test is to provide a rich source of stimuli to prompt the patient to "talk about himself," this is not the most significant use of these stimuli with aphasics. The pictures elicit a sample of the language of the aphasic which is often lacking in many other types of instruments. The rambling of the pragmatic aphasic, the substitution of neologisms for semantic words of the semantic aphasic, and the telegraphic language of the syntactic aphasic, are often clearly evident. So the test may provide both personality data and a rich sample of the patient's residual language.

The Projective Drawings consist of having the patient draw a house, a tree, a person, and then a second person of the opposite sex. The house and the tree may be eliminated and free drawings may be included, as desired. As in the case of the Bender Gestalt, these drawings may indicate the presence of severe levels of regression, organic brain pathology, and social-emotional adjustment problems. The male figure may be used in arriving at a mental age, using the norms provided in the Goodenough Draw-a-Man Test.[16] The examiner has found a fair number of aphasic patients who have drawn the head well above the body on personal drawings, indicating the patient's belief that his aphasia involved a literal "loss of the mind"--a departure of the mind from the body. Again, as in the case of the Bender Gestalt drawings, every effort should be made to get the patient to cooperate, for these drawings can contribute a wealth of information to a differential diagnosis.

Tests of educational achievement----While aphasia is primarily a speech and language problem, the problems of aphasics include disabilities in reading, writing, spelling, arithmetic, and other subjects taught in school. Obviously the typical academic achievement tests would be most useful in assessing the nature and extent of these dysfunctions. While there are many tests available for this type of measurement, two of the most useful are the Wide Range Achievement Test[17] and the California Achievement Test.[18] The Wide Range Achievement Test is simply and quickly administered and provides screening tasks from the simplest to very complex levels of achievement in Reading, Spelling, and Arithmetic. The test is standardized so that it provides age norms, grade norms, and quotients similar to the intelligence quotient, and thus the norms are most broadly applicable. The California Achievement Test is one of the more thorough and diagnostically significant of the usual public school tests and it may afford data to be compared with those data found in the patient's cumulative records of his public school achievement.

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[16] Florence L. Goodenough, Measurement of Intelligence by Drawing (Chicago: World Book Company, 1954).

Tests of vocational interest and aptitude----These tests, and those already described, may be used with mildly involved patients to assist them in readjusting to self-supporting, independent living in the community. Some of these tests, notably the Kuder Vocational Preference Inventory,[19] also provide data which may be most useful in planning treatment. For example if the patient rates highly in the "Outdoor" preferences and low in the "Scientific" preferences, his therapist should consider these facts in his selection of therapeutic stimuli.

The complete social and medical history of the patient should be available to the speech and language therapist, and results of the speech and language therapist's studies should be made available to the physician and the social worker. Of particular importance in planning speech and language rehabilitation is information concerning the patient's (1) onset of symptoms and the care and treatment he has received previously; (2) handedness; (3) educational-vocational achievement; (4) pre-morbid interests, hobbies, and avocations; (5) personal, social, and family relationships; and (6) problems growing out of his present life situation. This information is not only useful in planning the patient's rehabilitation, but is imperative in understanding what he is trying to communicate to those working with him.

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[17] Joseph Jastak, Wide Range Achievement Test (Wilmington, Delaware: C. L. Story Company, 1946).

[18] The California Achievement Tests are published by the California Test Bureau, Del Monte Research Park, Monterey, California; the 1957 edition is currently in use.

[19] G. Frederic Kuder, Kuder Preference Record (Vocational), Form CH (Chicago: Science Research Associates, 1948).

When all diagnostic studies are completed and some early exploratory treatment has been given, all available data should be used to formulate an over-all plan for the patient's ultimate rehabilitation and a formally structured treatment program may then be developed.

EXAMINATION OF PURPOSEFUL MOVEMENT

Harold Goodglass

From time to time, we encounter an aphasic patient who succeeds in circumventing his speech defect to communicate information effectively by pantomime. In general, however, one is disappointed to find that the loss of speech usually brings with it a great impoverishment of expression by means of gesture and pantomime. On turning to the literature, we find that the question has been treated in a very fuzzy way, from the writings of Hughlings Jackson down to the present. Some, like Jackson, regard the impairment of communicative movements as one of the elements of aphasia, with the implication that some process central to any act of communication is behind both the speech defect and the gestural defect. This position is typified by the suggestion of the German neurologist, Finkelnburg, that the problem is really one of "asymbolia"--a loss of capacity to use symbols in any modality.

From another point of view, the disturbance of pantomime and gesture has nothing to do with the processes of spoken language, but is merely evidence of an apraxia--a disturbance of purposeful movement. As you undoubtedly know, apraxia has been observed and studied both in patients with aphasia and without. It is classified as one of the disorders related to aphasia, in the sense that it entails a disruption of high order functions through injury to the left cerebral hemisphere. Moreover, the standard examination for apraxia includes the testing of pretended actions, both gesture and pantomime (a distinction which I shall shortly clarify). There are several types of apraxia, some with evidence for the good anatomic localization of the lesion involved. Liepmann, the German neurologist, studied these disorders from the point of view of their total independence, as psychological processes, from aphasic speech disorders. There is still another point of view with respect to the nature of gestural deficiency: namely, that we are dealing with an intellectual defect which is neither linked specifically to language nor to movement. You recall the Goldsteinian principle of abstract behavior; that the ability to pretend something which is not in the context of present reality is an abstract act. It can therefore be predicted that the ability to carry out pretended actions will be defective in brain injured persons who have lost some of their capacity for abstract behavior. It is clear, however, that this interpretation is applicable to non-aphasics as well as to aphasics.

With these possibilities in mind, my associate Edith Kaplan and I undertook to study the disturbance of pantomime and gesture in aphasics four and a half years ago. Our preconception was that there is a central process operating in the formulation of any information into communicable form whether by speech or by gesture. We realized that in order to establish this position, we would have to show that pretended action is impaired in direct relation to the severity of aphasia. Further, we realized that in order to differentiate this disorder from apraxia, we would have to show that the ability to carry out movements to imitation was not significantly impaired.

The basic methodological requirements for such a study are pretty obvious, and were fulfilled: namely, the selection of aphasic and non-aphasic brain injured subjects, at least roughly equated for age and post morbid Performance IQ, and a method for rating the severity of aphasia, in the aphasic group. The most difficult and novel part of the method, however, is the procedure for testing the ability to carry out gesture and pantomime. Let us consider the psychological nature of the task involved.

There are many kinds of communicative movements. Gesticulations, mannerisms, movements of approach and avoidance, are legitimate and fruitful subjects for study under the heading of nonverbal communication. However, we specifically excluded this type of activity from the scope of our study, because we wished to work with a standard set of expected responses. Confining ourselves to movements which convey an intended specific message, we made the following distinction between gestures and pantomimes, based essentially on the conventionalization of the movement:

> A gesture is a relatively standardized movement whose meaning is well known in the community; a pantomime is a movement which is improvised by the user to convey information about a thing, person, or action by some representation of form, action, or both.

With the category of gestures, there are some which seem to be vivid action pictures of the message they convey--for example, covering the ears to indicate too much noise; rubbing the stomach to signify a good meal; raising the hand palm out to indicate "stop." We called these Natural Expression Gestures. Others have lost any pictorial quality which they once had, but are recognized because of their high level of conventionality. We called these Conventional Gestures, and they include such movements as waving goodbye, saluting, indicating thumbs down.

The pantomimes, in turn, ranged in complexity from easily picturable objects or actions to complex narratives. A simple pantomime would be the request: "Show me how you would pretend to hammer a nail." Another, presented nonverbally, was to have the patient identify by pantomime a picture of an object which the examiner held up to him, such as a telephone or a spoon. Since I will demonstrate the examination procedure to you, we will omit details on the test items here. Suffice it to say that each item was first requested of the subject, then performed by the examiner for the patient to demonstrate his comprehension and to attempt to imitate. The complex narrative pantomimes were first performed by the examiner to test the patient's comprehension of them, to reduce the difficulty of the task, and to reduce the burden on the patient's auditory comprehension, since the patient would be asked to do what he had just seen demonstrated.

How can one assume that the examiner's rating of a patient's performance would agree with another's? While the gestures are fairly well standardized movements, the pantomimes are not and different people may choose to take different approaches in responding to a given item. We obviously could not legislate exactly how a patient should act out a "telephone" if he wished to receive credit for that particular item. For example, one person might limit himself to picking up the receiver and bringing it to his ear--a movement which almost has the status of a conventional gesture; another might include dialing a call, but omit listening to the receiver. The standards for scoring were defined in terms of the examiner's judgment of adequacy, and only a four level discrimination was asked: (1) performance is within the range of normal, making allowance for awkwardness of the non-preferred hand in hemiplegic individuals; (2) performance is relevant and recognizable, but with some obvious inadequacy; examiner can detect an element of relevance, in the movement, but it would have no communicative value to one who did not know what idea was intended; (4) totally inadequate movement or failure to attempt.

In order to test the reliability of such a subjective sounding scale, we took motion pictures of a series of sixty pantomimes done by three patients and showed them to a group of five judges, each of whom had to rate each item independently. A reliability coefficient in the low eighties--a bit low, but not totally unacceptable for individual clinical tests--was obtained.

Looking at the results from testing an aphasic group and a non-aphasic brain injured group, we found it necessary to give up our initial preconception about the nature of the disorder. I will try to make clear the evidence and our reasoning. First, there is a very marked difference between the aphasic and the non-aphasic groups in their ability to carry out gestures and pantomimes, with the aphasics markedly inferior in all IQ levels. Performance did indeed vary directly with IQ, but the lowest IQ group of non-aphasics exceeded the performance of the highest IQ group of aphasics. Second, the severity of the aphasia has little relationship to the degree of impairment in gesture and pantomime, within the aphasic group. Third, the opportunity to imitate brought only limited improvement in the aphasics' performance of those items which they had failed on simple oral command. The improvement rate was twice as great for the non-aphasics as for the aphasics.

Taking the last observation first, the persistence of difficulty even during imitation is a characteristic of apraxic difficulties and does not seem to be compatible with the concept of a difficulty at the level of formulating units for communication. Since most reported descriptions of apractic cases indicate left cerebral lesions, it is entirely to be expected that apraxia should occur in conjunction with aphasia, which also involves left hemisphere lesions over 90% of the time. It is also to be expected that such a relationship--based on anatomical contiguity of a gross kind--need not include much correlation in severity.

In everyday language, we can say that the overwhelming proportion of aphasics have some degree of ineptness for carrying out gestures and pantomimes; a few are exempt. A severe aphasic may have only slight difficulty and a mild aphasic may have severe difficulty.

We are now in a position to make some observations about the order of difficulty of various gestural tasks. First, and not surprisingly, the more conventionalized movements--those classified as gestures--are more readily carried out than the pantomimes. In examining a patient, then, we expect to find that he may be able to wave goodbye, pretend to applaud, or to beckon someone to come over, while he is unable to pretend to comb his hair, drink from a pretended glass, or brush his teeth. On the other hand, the Natural Expressive Gestures were not easier or harder than the seemingly arbitrary Conventional Gestures. The point being that the familiarity and constant repetition of the movement is the determinant of its preservation, rather than whether it is highly pictorial or not.

What about the problem of auditory comprehension? It is certainly clear that the patient who does not perform because he does not understand, must be distinguished from the one who cannot organize his movements. Our original procedure was heavily dependent on oral instruction, and we were forced to take precautions to avoid having the results determined by poor auditory comprehension on the part of the subjects. In revising our procedure for future use, we have introduced pictorial stimuli wherever possible. We have had stimulus pictures drawn so that the intended gesture or object or action to be represented in pantomime is clear, yet the precise position to be assumed cannot be copied from the picture. To elicit a pantomime of praying, for example, the picture shows a child from behind, kneeling at her bed, so that her hands are not visible.

One of the striking observations made by Mrs. Kaplan is that this population showed a form of pantomime which is highly characteristic of young children and which disappears late in childhood. This is the use of hand or fingers as though they were the pretended object. For example, the patient pretending to use a hammer, pounds with his clenched fist; pretending to brush his teeth, he rubs them with his index finger. This feature, which we termed Body Part used as Object, is most frequent in patients whose over-all adequacy is low.

We now believe that what we have been studying are high level disorders of purposeful movement which are properly regarded as apraxic. The term "apraxia" covers a number of analogous but somewhat independent defects in which the common feature is a disturbance of purposeful movement resulting from a cerebral lesion, but without paralysis or incoordination of the parts involved. There is an obvious analogy with aphasia--especially since an injury of the major hemisphere commonly produces bilateral symptoms of apraxia. The distinction from aphasia is that the movements involved do not in themselves constitute symbols of a linguistic system. Apraxias may be generalized or confined to certain parts of the body. The parts which may show localized impairment are the eyelids, the oral and respiratory apparatus, the upper limbs, and the lower limbs. There are well documented cases of severe apraxia confined to the non-preferred hand and these are always associated with injuries, which partially disconnect one half of the brain from the other.

As with virtually all organic psychological defects, the more a task is overdetermined by familiar contextual cues, the easier it is. The more it relies on the ability to reconstruct a movement from memory, the more difficult. Thus, the patient who has no trouble manipulating common objects may fail badly when he is asked to imitate the pretended use of objects, and do worse yet on simple oral requests to carry out pretended movements.

We are now continuing the study of gesture and pantomime in the context of a comprehensive examination for apraxia. Not only do we include a sampling of movements of the separate body parts which may be affected independently, but left and right limbs are also examined separately.

Here is an abbreviated version of the test, showing two items from each category.

A. Facial - Oral - Respiratory

   sniff
   blow out match
   close each eye
   wrinkle nose
   lick lips

B. Action--with Objects

   1. Hand to face
      toothbrush
      comb hair

   2. Hand away from body
      pencil
      saw

   3. Two-handed
      break an egg into bowl

C. Action--without Objects

   1. Conventional and Natural Gestures
      salute
      wave goodbye

   2. Pantomimes
      swimming
      sleeping

   3. Lower Limb
      grind out cigarette
      pedal bicycle

D. Comprehension--Simple Actions

   winding watch
   digging

E. Comprehension--Complex Pantomime

   picture hanging sequence
   cigarette lighting sequence

F. Performance--Complex Pantomime

picture hanging sequence
cigarette lighting sequence

THE TREATMENT
OF THE
APHASIC ADULT

111-112

COMPREHENSIVE REHABILITATION

Corbett Reedy, J. Hamilton Allan, Roy M. Hoover,
Harold Goodglass, Jon Eisenson, Paul Breeding

Mr. Reedy: We welcome this opportunity to express the interest of the Vocational Rehabilitation Administration in this workshop. As Dr. Burr announced in the beginning, we are co-sponsors, and actually our co-sponsorship was of the most modest kind. Our role has been more that of a participant because at one time or another all of the members of our regional office staff have had the opportunity to audit sessions of the workshop and it has been most educational to us. We must say to Dr. Burr that she has more than justified our expectations in terms of the quality of the workshop. In addition to being such a good professional leader, all of us are most impressed and grateful for the kind of hostess she has been, too. I think we would have been amply paid for this visit here just by the daily professional sessions, but the evening sessions have really put the "frosting on the cake."

Our subject this afternoon is Comprehensive Rehabilitation for the Aphasic Adult. I want to start off by asking our expert on definition, Dr. Eisenson, is _aphasic_ a noun or adjective? (Dr. Eisenson: _Aphasic_, I think, is an adjective.) Then with that bit of expert advice, I am going to be a little more careful to speak of these people as disabled persons with aphasia. (Dr. Eisenson: An adjective may be used as a noun, or any noun as an adjective!) I am aware that we take all kind of liberties with our language. (Dr. Eisenson: _Aphasic_ is a noun except when it is used as an adjective!) One of the things I enjoyed so much about the first session when Dr. Eisenson spoke was that he relieved me of all fears that this was going to be so stiff, and formal, and technical that one would have trouble just sitting through it. Some years ago we read a very interesting paper written by Dr. George Deaver, whom many of you know to be one of the real pioneers in medical rehabilitation in this country, in which paper he attempted to classify disabilities in terms of their relative handicapping effects on individuals. Now anybody, of course, who undertakes this task will run into all kinds of problems, but this is how I recall the classifications of disabilities that Dr. Deaver gave. He stated that from the standpoint of vocational and social efficiency the loss of hands is the most severe loss that one can experience. Secondly, he listed loss of locomotion; if you are bound to one place in today's world, you are severely restricted. Thirdly, he listed the loss of ability to communicate. Of course, you can scramble these around any way you want to, but I mention them only to point out the high recognition that loss of communicative ability, and the loss of ability to translate the results of communication into meaningful action, has in the general field of rehabilitation. Presently, we in VRA are placing tremendous emphasis on the whole area--the loss of speech, the loss of hearing, and all of these associated problems around the field of communication. They are receiving a great deal of attention in our service programs, in our research program, and in our teaching program.

This afternoon we get down to the real heart of the subject of the conference: Comprehensive Rehabilitation for the Aphasic Adult. Such words as restoration, reactivation, re-education, development, and so forth, come to mind as we think of the term of rehabilitation. A definition that has always appealed to me a great deal goes something like this: rehabilitation for a disabled person may be conceived of as restoration or development of the ability, will, and opportunity for effective and satisfying living. This emphasizes participation, work that is both remunerative and satisfying--not just the restoration or development of ability, but the restoration or development of motivation to use it and the provision of opportunity to use it. If either is absent, then, of course, the result is very highly compromised. If we think of these in a general sense as rehabilitation goals, then they become very comprehensive. If we put the condition of aphasia in proper perspective, we see that it is one of a constellation of physical, mental, and emotional problems, each one being of varying severity depending upon the individual and the circumstances. Thus, we may conclude that the problems of the aphasic individual are also comprehensive. If the rehabilitation goals are comprehensive, and the problem with which we are dealing comprehensive, then rehabilitation itself must be comprehensive when we think of the aphasic adult.

Now we are going to proceed to the discussion by our panelists. Dr. Allan is going to begin by explaining the services to the patient at the point at which he will first come to the attention of any of those who will work with him, and that is at the hospital level. Dr. Hoover will then discuss the medical management of the aphasic adult who is undergoing comprehensive services in a vocational rehabilitation center setting, such as Woodrow Wilson. Dr. Goodglass will explain the psychological aspects of the adult aphasic problem and the role of the clinical psychologist during the rehabilitation process, and Dr. Eisenson will discuss the contributions of the speech pathologist. Finally, Mr. Breeding, our vocational rehabilitation generalist, will discuss the specific services of vocational rehabilitation--ending, we hope, in the successful vocational adjustment of the aphasic individual. We will begin with Dr. Allan's presentation and then follow in order.

Role of the Physician:
Primary Phase of Recovery

Dr. Allan: Whenever aphasia is one of the presenting symptoms in a patient brought to the hospital emergency room for care, the immediate reaction of the examining physician is that there must be a lesion cutting off the blood supply and thus causing injury to those areas of the brain concerned with the complex problems of language function.

Damage to these areas occurs most frequently with: (1) severe head injuries causing contusion of the brain and intracranial hemorrhage; (2) strokes caused by atherosclerosis with superimposed thrombosis; (3) hypertensive hemorrhage; (4) space occupying lesion such as brain tumors.

Not long ago a man came into the office of the Vocational Rehabilitation Counselor here at Woodrow Wilson Rehabilitation Center and complained that he could not get a job. "Why not?" asked the counselor.. "Well," replied the man, "even if I could sing like Sinatra, dance like Astaire, think like Einstein, I still couldn't find a job." "But why not?" repeated the counselor. "Well," said the man, "who on earth could possibly afford me?"

At this point I cannot afford to sail under false colors and pretend to know anything about an organ as complex as the brain, because I am an Orthopedist, accustomed to simple mechanical problems. However, I am interested in rehabilitation. Our modern concept is to start rehabilitation when the patient enters the hospital. If we do this, I can share with you the layman's objective view of the entering patient and tell you some of the medical and surgical approaches and developments which have significant implication in the treatment of aphasia.

Head Injuries----First of all, let us consider head injuries which I am afraid will always be with us, due to the increasing number of highway accidents. In head injuries, the ultimate mortality and morbidity depend upon the quality of initial care. Lack of oxygen to the brain being the most usual cause of death, the maintenance of an adequate airway has priority over all other treatment. A tracheostomy,with oxygen supplied through the tube, is frequently needed to support the breathing mechanism. Lack of oxygen to the brain can develop not only as a result of inadequate respiratory exchange, but also from swelling, or from an expanding mass within the rigid cranial box which reduces the circulation and thus reduces the oxygenation of the brain. The signs of increasing intracranial pressure require careful assessment and recording. For example, initial consciousness followed by unconsciousness suggest increasing compression by a swelling or hemorrhage, and the surgeon must operate to decompress the edema or control the hemorrhage. On the other hand, continued unconsciousness from the time of injury, deprives the examiner of the keystone of clinical evaluation which is the level of consciousness, and so other techniques of detection such as a ventriculogram, or an arteriogram, or even a cranial exploration may be necessary to establish whether an expanding lesion is present.

Extradural hemorrhage is often associated with fracture of the skull and is frequently due to laceration of the middle meningeal artery, or one of the dural sinuses. Surgical treatment to stop the bleeding and evacuate the clot will permit a complete cure.

The subdural hematoma causes changes similar to the extradural hemorrhage. It may be due to arterial bleeding. It may be chronic as a result of slow venous bleeding, and show a picture of slowly increasing pressure. In either case surgical evacuation of the clot relieves the pressure.

Those patients who are unconscious from the moment of impact, and whose condition deteriorates, are difficult to evaluate and hemorrhage may be present in any part of the brain. If there is a question of differential diagnosis between a cardiovascular accident, such as a stroke and a space consuming hematoma, a ventriculogram may be helpful. This is a diagnostic technique in which the ventricles of the brain are infiltrated with air. An X-ray is taken in order to determine whether the ventricular system is normal or is distorted. If it is normal, surgical attack is not indicated. If it is distorted and compressed, surgical attack is indicated to relieve the compression.

Perhaps the most promising development in treatment of head injuries is the procedure of hypothermia--that is cooling the body to subnormal temperatures. Trauma to the brain causes a marked rise in temperature which is an unfavorable prognostic sign. This increased temperature is associated with increased metabolic demands of the brain, which presumably is already suffering from inadequate circulation. If the temperature of the body is lowered, the metabolic needs of the brain can be met, even if the cerebral circulation is reduced by the effect of the injury. Treatment with hypothermia for days or weeks allows the patient with severe head injuries to survive, whereas previously they would have been considered hopeless. Patients have been maintained with body temperatures of 30 to 32 degrees centigrade for four to six weeks. However, a few days is usually all that is necessary.

At the University of Virginia Children's Rehabilitation Center now is a nine year old boy who was struck by a car seven months ago, and who suffered a depressed skull fracture. His condition was critical when he reached the hospital. He was taken to the operating room where the skull wound was cleaned and the depressed bone fragments pressing on his brain were elevated. Because of his critical condition he was placed on hypothermia in the recovery room and a temperature of 31.6 to 33.5 degrees was maintained for about a week. A tracheostomy was also done to improve his airway.

At the end of the week the signs of intracranial pressure were subsiding, so that the hypothermia could be discontinued. About three weeks later, he slowly began to recover consciousness. He was noted to have a left hemiplegia, bladder and bowel incontinence, a marked emotional disturbance, and an expressive and receptive aphasia. During his period of rehabilitation care, the aphasia cleared slowly and when tested six months after the injury, his reading was normal. Writing and arithmetic processes were still below par. His difficulty in naming objects had cleared. It was noted that occasionally his speech would be mumbling and nasal whenever he became very depressed. This child has continued to show progressive recovery and it is anticipated that he may recover from the aphasic complication completely.

I think it is quite probable that the use of hypothermia in this case reduced the metabolic needs of a brain whose circulation had been already placed in jeopardy by the injury. By this means this child was able to survive and some of the effects of intracranial pressure, such as the production of aphasia, have been reversed.

The Stroke Patient---- The next important cause of aphasia in a patient being admitted to a hospital is a stroke. This is the hallmark of all forms of vascular disease of the brain. This is clinically a focal neurological disorder which develops suddenly and is caused by interruption of the circulation to the appropriate area of the brain. Vascular disease of the brain can be put into two general categories: (1) ischemia which is, of course, due to lack of oxygen, and (2) hemorrhage.

Ischemia is the more common and accounts for 75% of the strokes. Brain tissue cannot store oxygen so it is dependent from minute to minute on an adequate flow of blood. If blood flow is cut off for more than a few minutes or when an artery is plugged by a thrombus or embolus, the tissue supplied by that blood vessel dies. By the 1950's we became aware that the basis for ischemia in the brain was the atherosclerosis of such arteries as the carotids, the vertebrals, and the basilar. Atherosclerosis refers to lining deposits on the inner blood vessel wall of yellowish plaques containing fatty material and cholesterol.

As you are well aware, the arteries of the brain and the arteries of the entire body are a reflection of man's lifelong experience. Atherosclerosis is a secondary disease which slowly narrows the vascular channel. As man goes through life he has battles with micro-organisms which damage the lining of the vessels. Hypercholesteremia, high blood pressure, obesity, heavy smoking, hypothyroidism, kidney damage, diabetes, physical inactivity, personality and psychological factors, hereditary predisposition are all factors which produce atherosclerosis and ischemia or softening of the blood vessel wall and hemorrhage.

The medical problem, therefore, is to develop an early assault against atherosclerosis because aphasia, if it develops as a byproduct of a generalized arteriosclerosis, may reflect a major failure of medicine.

Our interest for this presentation is perhaps in the patient who has atherosclerosis and is becoming aphasic. In such cases, anticoagulant therapy may be indicated. Anticoagulants are drugs which prevent clotting and, therefore, inhibit occlusion and thrombosis. They add, however, the risk of increasing the danger of hemorrhage. Hence, anticoagulant therapy is very exacting therapy which must be carefully controlled. It has been helpful in reducing the number of intermittent transient recurrent strokes. It has been helpful in preventing the step-like progression type of stroke, in individuals who have normal blood pressure. It has lowered the death rate in these cases, also, by preventing the recurrence of blood clots in the major vessels of the lung. However, in those individuals who have had severe persisting strokes and whose neurological deficit has reached a stable plateau, it has not been helpful. In other words, it has no effect on the thrombus once it has formed to plug the vessel.

The dissolving of clots in a blood vessel depends upon fibrinolytic enzymes normally present in the body. The clinical use of such enzymes has not been investigated until recently. These enzymes have now been prepared in sufficiently pure form so they can be injected intravenously or into the vertebral or carotid arteries in the neck, to dissolve intravascular clots. The fibrinolytic enzyme "plasmin" has been used with anticoagulants in a double blind study with patients suffering from cerebral thrombosis. From the data obtained, this therapy has been helpful in some of the cases in which it has been used. Much more information is needed, however, before it can be put into general use.

Today there is considerable enthusiasm for a surgical approach to certain types of strokes. Stenosis or occlusion of the carotid and vertebral arteries plays a role in a large number of strokes, and it is in this location where surgical attack can be helpful.

Last month at the Scientific Assembly of the Medical Society of the District of Columbia, Dr. Michael Debakey, Professor of Surgery at Baylor University, reviewed the surgical techniques that have been used to remove obstruction or repair blocked arteries. These techniques include: (1) endarterectomy, which is opening the artery and reaming the artery and pulling out its clot; (2) patch grafting in which the diseased arery is split lengthwise, scraped clean of its debris, and covered with a patching material, a process likened to vulcanizing; (3) bypassing grafts of dacron cloth tubes.

Within the past ten years at Baylor University, a thousand patients have been operated upon to repair blocked arteries supplying the brain. Of these, 87% are alive and without symptoms of their strokes for periods now of two to ten years.

The development of arteriography is responsible for the success of these operations which were just a surgeon's dream in 1950. Arteriography is a technique whereby doctors can visualize constrictions of the blood waves. In this technique, solutions of a highly radio-opaque fluid are injected into the arterial network to be studied. X-raying the fluid, as it passes or fails to pass, produces a picture of the area of obstruction. With such localization, the site of the obstruction may be attacked surgically and cleaned out. When the symptomatic pattern of segmental occlusive disease, involving the extracranial portion of the major arteries which supply blood to the brain, is present, arteriography is done and is very helpful in localizing the site of the obstruction. (The symptomatic pattern of blockage of these arteries is roughly as follows: (1) Internal carotid artery occlusion is manifested by contralateral motor and sensory deficits, ipsilateral visual disturbances. (2) Vertebral artery occlusion by bilateral motor and sensory defects, bilateral visual disturbances, vertigo and diplopia. (3) Occlusion of the great vessels, subclavian and innominate, by a combination of patterns of neurological symptoms noted, with internal carotid and vertebral artery occlusion, plus ischemic arm symptoms.) As indicated previously, the anatomical and functional results have been quite satisfactory. The following case illustrates the circumstances under which surgical restoration of blood flow can be attempted.

A man, 52 years of age, sustained a stroke one month before admission. There was right hemiplegia with aphasia. He was treated promptly with anticoagulants and had almost completely recovered. When admitted to the hospital, arteriography disclosed stenosis of the left internal carotid artery at its origin and occlusion of the left subclavian. The extracranial and intracranial vessels on the right were normal. Endarterectomies of the left internal carotid and the left subclavian were performed, under general anesthesia, with restoration of blood flow. The patient's recovery was uneventful and he has remained asymptomatic since then.

Tumors or Space Taking Lesions----I would like to illustrate, with a case history, the role of the brain tumor as a cause of aphasia and its surgical management.

A 69 year old lady was admitted to the hospital with the history of suddenly becoming confused. She was unable to remember people or dates. Coincidental with her confusion and memory difficulties, the patient noted that she had begun to have difficulty in expressing herself during conversations but claimed that she always knew what she wanted to say. Furthermore, she was unable to identify objects placed before her. There was no headache, nausea, vomiting, visual disturbance, or localizing motor signs. The neurologist examined her and noted that she had a marked expressive, with much less receptive, dysphasia. From his examination he felt that she had a left hemispherical spacetaking lesion, probably a neoplasm. The left carotid arteriogram revealed highly suggestive evidence of a mass in the temporal lobe, predominantly posterior. The ventriculogram which was done was not satisfactory, but the surgeon went ahead and did a left craniotomy, and found a well circumscribed tumor present on the surface of the temporal lobe. This was completely excised. By the fifth post-operative day, the patient's expressive aphasia had improved and she was able to recognize familiar objects and call these objects by name without difficulty.

In summary, let me say that in consideration of the medical management of the early aphasic, it must be remembered that in cerebrovascular disease, damage to brain tissue is liable to be irreversible. Therapy of all kinds--dietary, hypotensive, antidiabetic, anticoagulant, perhaps fibrinolytic and surgical--must be used at the earliest stage to halt disaster. I would like to leave you with the thought that in these cases, early treatment is a matter of such urgency that in every community hospital there should be a special cerebral vascular receiving unit to which patients may be referred for diagnostic and emergency management.

Role of the Physician:

Secondary Phase of Recovery

Dr. Hoover: As an introductory statement, I wish to disclaim any considerable amount of certain knowledge about aphasia. Any such knowledge I may have is related to the observation of these cases as they have been treated and followed through the phases of their rehabilitation.

Over the years we have seen many individuals with aphasia. These cases as seen in a comprehensive rehabilitation center are those having residual impairments of the pathological conditions described by Dr. Allan. We are dealing with the residual physical impairments, including aphasia, resulting from their brain damage. These residuals, in addition to aphasia, frequently include hemiplegia or other paralysis or incoordination interfering with bodily function.

As we observe these individuals through the various phases of physical and occupational therapy--bracing, surgery and all of the other steps necessary in adjusting to life and society and, hopefully, to employment--there seems to be a direct relation between their progress in the use of language and their improvement or lack of improvement in physical status. On entrance to the Center, they have passed the acute phase of the condition and are ready to enter speech therapy, physical therapy and occupational therapy; in order to develop to the maximum their potential for function as normal individuals in normal society. If they are unable to walk, they are taught walking with the help of mechanical aids--such as parallel bars, canes or braces. If there is a hemiplegia and only one upper extremity is normal, much time and work is concentrated on an attempt to develop the useful function in the paralyzed hand and arm. As they are observed in therapy, there appears to be a definite parallelism between the improvement in language use and that in ambulation, self-care, and the activities of daily living.

There is a wide variation in the amount of improvement secured in different types of brain damage cases. In discussing this phase of the problem, I will start with that type of case which to me has the poorest prognosis: the adult cerebral palsy case with severe speech difficulty. The prognosis is poor for material improvement in ambulation or use of the hands, and I do not recall one who has made any marked improvement in speech. These cases are at the very bottom so far as expectations are concerned; though I always hope that I am mistaken.

The second group includes so-called "stroke" cases. These are divided into two distinct sub-groups: first, the elderly individual with generalized arterial sclerosis, hypertension, and the other degenerative conditions which frequently are combined, and second, the young individual, frequently in the twenties or even younger, whose cerebral vascular accident is the result of rupture of a congenital aneurysm of a cerebral vessel. These later cases have a much greater probability of extensive improvement in walking, use of the affected hand, and improvement in speech than the person with generalized degenerative disease. The outlook for eventual employment of these younger individuals is reasonably good.

The cases that have, in general, the best prognosis are those due to trauma and those due to brain tumors. The results in such cases are usually either quite good or very bad; few of the latter type ever reach a rehabilitation center, for obvious reasons. The ones that learn to walk well, regain use of the hands for dextrous activities, self-care, and the activities of daily living, almost always make very marked improvement in their language abilities, and thus have real vocational possibilities.

I believe that one case history will demonstrate these facts better than any amount of discussion. A number of years ago, a young man was referred to the Center by Vocational Rehabilitation of Texas. Inevitably, he was known as "Tex." He had completed two years of college before his injury. Four years before his admission to the Center, he had been involved in an airplane crash; in this crash he not only sustained severe

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brain damage but a compound comminuted fracture of one femur. He had been unconscious for a period of six weeks. At the end of that time, when consciousness returned, he was completely aphasic. He was unable to speak, he could not understand the spoken language, he could not write, and he could not read. At the time he entered the Center, he could speak a little but he still was unable to either read or write. The leg had healed with 3 1/2 inches of shortening and with sufficient external rotation that his knee motion was at right angles to the motion of his hip, and he was unable to take more than a few steps. He was entered in physical therapy, occupational therapy, and speech therapy. It was obvious, in a very short time, that he would never be able to walk well without surgical correction of the serious mechanical difficulties of his leg. The first surgical procedure was an osteotomy of the femur, rotating the distal fragment around so that the knee operated in the same plane as the hip. By this means, all parts of the leg functioned in one plane instead of the hip going in one direction and the knee and foot at a right angle to it. The heel cord had to be lengthened, because there was a contracture that made it impossible for him to stand on the bottom of his foot. In addition, there was a division of part of the nerve supply to the calf muscles, with stabilization of the foot being carried out. It was quite interesting to observe that as each of these procedures was accomplished, he came nearer and nearer to the ability of normal walking and the remainder of his picture improved. He became quite interested in painting, which was done with his left hand, though he had originally been right-handed. I have on my study wall an attractive, typical Texas scene of a calf branding that he painted for me. His speech improved to the point that he could tell a good story from beginning to end, without stopping for lack of words. He could write short sentences and was able to write words perfectly well from dictation. At the end of a year when he left us, he could recognize printed words, but was still unable to read a sentence. Followup information disclosed that he eventually learned to read quite well and, in fact, spent much time in reading. He opened a store in the country down in Texas and considers it ideally located, as it is right on the river bank. He has a bell arranged on the door of his store that will ring when a customer comes in, so he can spend most of his time on the river bank fishing and reading and only come to the store when he has something to do. He is also pleased with his clientele because there are not many customers. Those that do come, buy large orders which are to last for weeks or months and thus he is able to spend as much time as he wants reading and fishing. This case is an excellent illustration of the progressive improvement in speech, ambulation, and ability to carry out the ordinary activities of daily living and self-care. It has also impressed on me the fact that speech patterns are not re-learned in a short time. This man left us five years after his accident and yet it was a period of about two years before he was able to read and speak normally. It has been interesting to watch the parallelism between the improvement in speech and physical ability in our cases here at Woodrow Wilson Rehabilitation Center.

Role of the Clinical Psychologist

Dr. Goodglass: From the point of view of the clinical psychologist, the first step in the treatment of an aphasic is to evaluate the problem and set appropriate goals. I have deliberately chosen the non-committal term "treatment" in preference to "rehabilitation," which, in reference to adult aphasics is loaded with an implied promise of more than is often within our power.

In outlining what goes into the psychologist's evaluation, I have decided not to worry about precise lines of professional authority. In some situations, there is no psychologist. The neurologist, the speech therapist, the social worker, may well be covering parts of the same territory--and indeed should be doing so.

Given the medical and physical limitations of an adult aphasic, there are still three more general areas of information which must be assembled and have their blanks filled in, before an adequate psychological analysis is possible.

The first phase of the evaluation is the social and personal background.

What sort of role was the patient playing in the world of work and family, and what was his psychological equipment? The raw data contributing to this picture is, in part, covered by the questions which are often listed on the fact sheet of a test record: age, education, marital status, occupation, language spoken, hobbies. The more complete the background picture, the less likely that there will be misinterpretations or gaps in the understanding of the present situation. For example, we cannot even evaluate the extent of the aphasic problem, unless we know the level of premorbid language accomplishment. How unrealistic it would be to undertake therapy of a severe spelling defect in a patient who turns out to have gone to special classes in elementary school because he could not learn to read and spell. We cannot evaluate the significance of the present illness in the patient's life unless we know the level at which he was functioning. How central were verbal and symbolic skills to his way of life? For example, if he was an active professional person, who is not expected to resume his professional career, it is very important to know whether other satisfactions in life also contributed to his self esteem.

The second major area of psychological assessment is the current status of psychological abilities and deficits. The examination as carried out by the psychologist has multiple goals. The immediate result of the examination is a full delineation of the patient's status with respect to various cognitive, perceptual, and problem solving skills. These are measured by a battery of intelligence tests, language performance tests, and tests of special organic deficits. Not all of this data can be turned to use in the rehabilitative process, since the test samples many performances which rarely, if ever, enter directly into coping with the requirements of daily life and work. The psychological examination reveals many deficits which the patient is unaware of, and which do not become a focus of rehabilitative efforts. For example, in the course of the examination, it may develop that the patient has a striking, severe inability to understand words spelled orally to him. While this is a significant component of his aphasia from the point of view of understanding the total defect, it is difficult to think of many occupations where this would be a handicap. It is up to the clinical psychologist to interpret the functional strengths and handicaps emerging from his examination, recognizing squarely that this requires a special interpretive effort.

The psychological examination provides an important yardstick of progress during the period of recovery since part or all of it may be repeated serially. Finally, the psychological examination is a basis for research, insofar as it may serve to reveal regularities that improve our understanding and our ability to predict from one set of findings what other important findings will be.

The aphasic is, by definition, a brain-injured person. The presumption is that he has suffered some degree of loss in intellectual ability. The Wechsler Intelligence Scale provides a sampling of performances by which we can judge the intellectual efficiency. We know that we can measure intelligence only by the results it produces, and that the loss of language generally deprives us of the verbal means of measurement, and forces us to rely on the Performance IQ of the Wechsler. Suppose, however, that the patient has also suffered a specific impairment in his ability to execute visual-spatial tasks, such as those involved directly in three of the five performance subtests. His Performance IQ may then also be spuriously low and it is up to the psychologist to make the determination of the validity of the numerical test score. When the test appears invalid, it is up to the psychologist to make an estimate, on the basis of qualitative cues, such as the patient's speed in grasping new situations, his self-critical capacity, and his performance in isolated instances.

The evaluation of the aphasic patient extends to a survey of those higher perceptual motor and symbolic skills on the periphery of language, which are susceptible to specific damage from left cerebral injuries. Thus, in addition to a study of the nature of the patient's changed language functions, the psychological examiner studies the patient's directional orientation, sense of left versus right, visual-spatial problem solving ability, calculation, and capacity for carrying out purposeful movements, both with real and pretended objects.

One of the benefits of a complete and analytic assessment of the patient's psychological status is that what passes for confusion or apathy may, on adequate examination, be revealed as a specific impairment, and the way be opened to useful communication, and to an entirely changed outlook on the patient as a functioning human being. At our unit, this has proved several times to be the case with patients who were apraxic and consequently responded poorly to verbal command, as well as seeming conspicuously lacking in initiative to carry out movements on their own.

The third phase of the evaluation is the concern with the patient's emotional contact with his situation. Here the psychologist must attempt to distinguish between premorbidly existing adaptive mechanisms and evidences of pathological change and restriction. For example, we are given an aphasic patient who is unusually cheerful and good-natured in the early, severe phase of his illness, and accepting of the invalid's role. The question arises as to whether this is merely an intensification of a pre-existing passive adaptation, with denial of the anxiety and depression normally associated with loss of function. If this is the case, the psychologist may well find intellectual functions relatively intact. Perhaps the patient has enough residual speech to be able to take the Rorschach test; perhaps he has enough skill in handling a pencil to be able to reveal, by his Human Figure Drawing, something of his long-standing personality predispositions. If he is able to cooperate with these projective tests, there will be more information available to guide us in understanding his true motivational state. Given the circumstance of a euphoric, agreeable patient, another set of test findings may convince the psychologist that this patient has suffered a lasting change in his capacity to appreciate what is socially appropriate, and a loss of concern for socially meaningful goals, with an accompanying superficiality of emotional response and a chronic air of silliness. Here again, we see how important the information on premorbid personality is, to the understanding of a patient's current behavior.

We can now get a more concrete idea of how the psychologist can contribute to the setting of goals on initial evaluation. In the case of the first euphoric patient, he may suggest that our sights be set fairly high. He may recommend that the patient be led gently to assume more responsibility in his life, and that the family be encouraged to lead him in the same direction, within the bounds of reality. He may suggest that personnel anticipate a depressive reaction later on, and he may conduct some supportive therapeutic interviews, if this seems necessary in the course of treatment. Individual therapy is not necessarily needed, nor practical in terms of time. Most often, it is the language therapist who has the most intense and influential relationship with the patient. The psychologist can contribute most effectively by consulting with the therapists on the management of the patient so as to maximize his motivation and to keep the treatment program geared to the most realistic assessment possible of the patient's capabilities.

Role of the Speech Pathologist

Dr. Eisenson: Let me say at the outset that I think one of the important roles of the speech pathologist is to understand the psychologist--to understand everything he says to you. In many instances a properly trained speech pathologist is not only able to understand what the psychologist has to say to him, but may himself be able to obtain that information--if he has the proper credentials, the proper training, the proper certification, and can cross union lines because he holds more than one union card. But if he does not he had better be careful!

The role of the therapist in the early stages of recovery is to be available to the patient, so that the patient appreciates that should he need help it will be there for him. A good therapist is not an interfering therapist. A therapist should always take the position that he should never get in the way of recovery. He stands by and lets it take place. He does not deceive himself that recovery is taking place because he is present. He merely acknowledges that it is taking place. It is only when there is a reduction of evidence that spontaneous improvement is taking place that the speech therapist needs actively to get into the act. I do not think the patient should be exposed to a therapist who has a box fitted with equipment and who thinks that the patient is going to be trained in terms of what happens to be in that little equipment box. This is not the role of the therapist in the early stages, and from my point of view, is not the role of the therapist at any stage. In psychotherapy there have been one or two studies to indicate that as therapy proceeds the patient begins to reflect the personality traits, the values, and attitudes, of the therapist. The longer the patient is in therapy with a particular therapist the more the two resemble one another. This may very well take place with aphasics, but I do not think it is necessarily a healthy thing to have happen.

Another important role for the speech pathologist is to be aware that a brain damaged patient is a patient with an electrical circuit that gets overloaded very easily, and if it gets overloaded it becomes defective very readily. Often the patient is in the position of the housewife who has all kinds of gadgets, but since the electric wiring in her house was not intended for all the gadgets for which she is now domestic engineer, she constantly blows fuses. After a while she knows where to put in a fuse to get the circuit working again, but she still puts on the radio while junior has the TV on, while she is ironing with a five thousand watt iron. Other things may also be happening in the house. Of course, the washing machine is going and the dryer may be working while mother is ironing and listening and junior is TV viewing. As a result, the electrical circuit is not very efficient and the housewife wonders why. The aphasic patient has a deficient electrical circuit. It gets overloaded easily. Any time you begin to overload a circuit, you are in danger of reducing efficiency of performance. This, of course, has implications for therapy. I think it is what Wepman had in mind a long time ago when he talked about "finding a circuit," and establishing a modality circuit for training. This is the approach, I think, that Mrs. Taylor is using when she directs clinicians, "Please be quiet," or she has a little sign that says, "Shut up please, don't talk so much, please!" We overload the circuit by all the verbiage. Stimulate through one modality. It is not always necessary to talk by word of mouth. Use the visual presentation, the visual modality, when you get correct response to the visual. I agree with Mrs. Taylor that we tend to talk too much. This is something that we must constantly guard against.

In the light of this general background, let me give you the kind of thing that everybody likes: a number of points. What kind of patients are we dealing with? Dr. Goodglass has told you what kind from the psychological view; Dr. Allan has told you what kind from the medical view. Here are my reminders: (1) Most aphasics, especially those with temperoparietal lesions, suffer from some degree of impairment in intellectual efficiency. Note the word efficiency. This means that only under optimum conditions do the patients work well. Under optimum conditions they sometimes can work very well. But optimum conditions are not normal conditions. Testing is usually done under relatively good conditions. A fine relationship is set up between the clinician and the patient; usually the room is fairly quiet and the ambient noise level is low, and there is very little distraction. You test and find that the patient does pretty well. He may come through with a rather good score on the Weschler, or whatever test of intelligence you use. Usually these tests of intelligence over-estimate the functional ability of the patient, because he has intellectual inefficiency. If you can create a setting in which he can work as easily, and with as good a relationship, as you do in good intelligence testing, then you can get the patient to work at his maximum. The implication of this observation is that we must help the patient to become prepared for those intrusions that reduce efficiency; we must build them up to this. Do the kind of thing that Van Riper talks about in his desensitization therapy. You cannot keep a patient isolated forever; but neither should you overload all at once. Find out what loading he can take, and then build up to it and hope that in time more and more loading can be taken by the patient, and that he will continue to work with relatively good efficiency. (2) Most aphasics manifest neuro-time lag. In severe forms, and under stress, this is expressed in perseveration. (3) Most aphasics, I believe, suffer from some degree of impairment in sequential behavior. They cannot deal with sequences very well, especially with ephemeral sequences--sequences that cannot be reproduced by taking a second look at the content. This is why the auditory approach is such a difficult one for the patient. He may have both auditory inefficiency and sequential difficulty. He cannot readily rehear what you have said, though he may be able to resee what he has failed to see and get the first time. (4) Most aphasics usually suffer from a degree of receptive involvement, greater than can usually be determined either in the taking of the inventory or in usual conversational situations. (5) Another point is that anxiety, feelings of inadequacy, and rapidly changing situations are productive of both perseveration and catastrophic behavior. Here again, we have indications of the result of overloading the capacity of the circuit. (6) Many aphasics strive too hard and aspire too high, at the outset. They tend to avoid simple language because of the desire to behave with premorbid linguistic proficiency. So, we ought to encourage them to give us whatever language is available to them, rather than insisting that they always give us the highest language that is most appropriate for the situation. If an aphasic gestures in a way to indicate very clearly that he means something by his gesture, this too is language. If, by any means at his disposal, he gets an idea across, this is the kind of behavior we should reinforce at the outset. If we reinforce the kind of language that he has available, we provide a basis from which the patient can operate and a foundation upon which we can begin to establish something. This is the kind of thing I think the Russians are doing so wonderfully well. At least they say they are doing wonderfully well and I take their word for it. This is the kind of thing that Luria is describing in his various writings. The important thing is to establish a second signal system. Language of some sort is a second signal system. It does not have to be the conventional language that you and I use. If there is a reliable, meaningful grunt, and if the same grunt consistently takes place in the same situation, accept it as having meaning and reinforce it. From that grunt on, the individual can begin to integrate--to organize his nervous system. All too often, what we are trying to do with the aphasic is to help him to organize his nervous system in the way we think a nervous system is normally organized, forgetting that he has an abnormal nervous system. If he could organize it the way we think it should be organized, what would we be doing in the act at all! The patient would not need us. He would be moving along very much on his own. (7) Most aphasics suffer from some degree of ego involvement. At the end of a training period with an aphasic I once said: "What was your problem at the beginning? Can you tell me so I can tell others about it?" He told me: "Let me draw you a picture of my problem." He drew a picture of himself coming into therapy at the beginning. He was weighed down by two satchels. One satchel he marked EGO and the other one, a tiny little bit of a one, he marked RESOURCES. His drawing showed him sweating and leaning towards the ground in the direction of the heavily loaded EGO satchel. He said: "This was I at the beginning. Loaded down by my ego and unable to use my resources. I was literally being dragged to the ground." I then asked: "Now draw a picture of yourself today?" He drew a second picture, and the EGO satchel was still the same weight as in the first drawing. The patient explained: "You know, out of habit I still walk this way." I thought that was a pretty good presentation of his problem. (8) Now, a final point is that the purpose of aphasia therapy is to make linguistic content, which is somehow not available to the patient, more readily available. Any technique for accomplishing this purpose that does not violate the ego and the personality of the patient is an acceptable technique. I probably ought to add, any technique that does not violate the ego and the personality of the clinician is also an acceptable technique.

Role of the Rehabilitation Counselor

Mr. Breeding: In discussing comprehensive rehabilitation services for the adult aphasic, I will use the term "rehabilitation" rather than "vocational rehabilitation," since a number of professional groups play significant roles in the total rehabilitation process. In recent years, there has been an increased awareness of the importance of social problems in rehabilitation. Factors in the disabled patient's personal life affect his response to medical treatment. These factors fall into many areas--such as economic, emotional, social, vocational, educational, and others--and workers from different fields are inevitably drawn into the treatment of the patient. The doctor, the social worker, the nurse, the physical therapist, the occupational therapist, the speech therapist, the psychologist, and the rehabilitation counselor are all dependent on each other in the rehabilitation of the aphasic adult.

If the doctor believes that the individual should not, or can not, resume his former work, a change in occupation may be recommended. Such a recommendation may mean leaving home for rehabilitation services. It may involve a change in economic and social status; a change in educational plans for children. It may induce in the patient severe feelings of hostility and guilt--hostility that this painful and bitter disaster has happened to him, and guilt that he is, in some way not understood, being punished for something he has done. Leaving home may mean that the patient, and his family, will urgently need help; help in filing applications to departments of Public Welfare; help in arranging for family housing; help in settling business matters; and help in accepting an altered way of life. Leaving home means separation from family and a familiar environment. In a rehabilitation center, the patient will live among strangers, where disability is paramount in all minds; where a minor event may become a crisis. The patient will be faced with many problems, and all persons caring for him must be aware of these problems and must deal with him accordingly. A long, careful period of preparation is often required before the patient can even begin a training program. He may be timid or resistant or concealing his fears. He may require supportive help. The rehabilitation of the aphasic may be a long drawn-out and trying process--for the patient, for his family, and for all who work with him. We must try to understand the aphasic and to see his problems through his eyes. We must be able to evaluate his readiness to accept, and help him to accept, any rehabilitation recommendations that may be made. Rehabilitation services are essential, but before the patient can use them he must be emotionally prepared, and he must have accepted the diagnosis of aphasia and/or hemiplegia. Comprehensive rehabilitation begins at the point of diagnosis, but the patient must accept the diagnosis, and the consequences of the diagnosis, before he can be vocationally rehabilitated.

Many such factors thus play a part in the patient's response to vocational rehabilitation. I remember the case of a 42 year old, successful radio announcer who suffered a cerebral thrombosis, severe paralysis on the right side, and loss of speech. He received medical treatment, speech therapy, and physical therapy, on an irregular basis, in his home community for about a year. At this time he was brought to the attention of a Vocational Rehabilitation counselor. Upon investigation, it was found that considerable improvement might be expected with intensive therapy. It was also found that his savings had been depleted, and that his family was in dire financial need. The reaction of this man and his family to our discussion of possibilities for further rehabilitation was tears and even some resentment. After numerous counseling sessions, arrangements were made to admit him to the Woodrow Wilson Rehabilitation Center, and help was given in filing an application for public welfare assistance. The patient stayed at the Center for about two months, and then left, with little improvement in his general condition. Later he was placed by the Vocational Rehabilitation counselor in sheltered employment in a Goodwill Industry, where he earned 35 dollars a week. He then applied for Social Security Disability Benefits, which were allowed after two months on this job. With this assured income, he quit the job. He now does household chores, while his wife works outside the home.

I also recall a successful case. This was a high school principal, in his late forties, who suffered a stroke during the last week of a school year. For weeks he was in critical condition. Immediately after the acute phase was past, however, the physician and family embarked on a program to restore his lost mobility and speech. Vocational Rehabilitation was asked to join in the long range planning. After thorough medical and vocational evaluation, it was concluded that intensive therapy and vocational training were needed. Plans were made for this patient to enter the Woodrow Wilson Rehabilitation Center, with Vocational Rehabilitation providing the full cost of physical therapy, speech therapy, and training in radio and television repair. After many months he recovered fully from the paralysis, developed some useful speech, and was qualified to work as a radio and television repairman. Today, he operates his own radio and television shop and leads a useful life in his community.

Our emphasis in Vocational Rehabilitation is on preparing disabled persons for employment. In our kind of economy and society, there are fewer and fewer jobs in which the ability to communicate effectively is not important.

I feel strongly, therefore, that more attention must be given to correcting or reducing impairments of speech among the disabled who are being served in our rehabilitation programs. Rehabilitation can not be a "piecemeal" affair. For the aphasic adult, it is a long and difficult process; it demands the patient understanding, and cooperative effort of each one of us.

Summary

Mr. Reedy: We have had a dramatic presentation of the course of rehabilitation that must be followed with the person suffering the condition that produces aphasia and related problems from onset to the point where he is functioning again in employment or in social living. While we have here only samples of the varied professional contributions that make up this process--medicine, speech therapy, psychology, vocational counseling--we all realize that many, many more professions may be concerned at various stages of this process. We are conceptualizing an ideal situation where all necessary services are available, and properly timed, and with a continuation of services until the maximum improvement has been achieved. One of the significant advances in rehabilitation is the development of specialized programs; the tailoring of a large number of specific rehabilitation services for treating specific conditions. For example, there exist very effective amputee rehabilitation programs and equally well developed programs for the cord injury case. Out of our intensive experience, study, and experimentation in recent years, we should now be able to fashion a rehabilitation process that is very specific to the condition with which we are dealing: aphasia.

LANGUAGE THERAPY

Martha Taylor

The notion of rehabilitating patients who have acquired aphasia was virtually unknown, at least on any large scale basis, until World War II. Before the early 1940's, an abundant literature referring to the neurological classification of aphasic impairment contained only an occasional and isolated reference to retraining. These accounts were largely clinical descriptions of single cases where attempts had been made to retrain patients in reading and writing skills. In addition, there were a number of descriptions of aphasic patients who recovered communication function through spontaneous recovery.

It was not surprising immediately following World War II to note a heightened interest in the possibility of retraining aphasic patients. There was during and after the war a considerable number of combat veterans who had acquired aphasia through traumatic war injuries. Despite the fact that there was really no history of aphasia rehabilitation, there was an immediate and urgent need for retraining. This need forced the creation of a new horizon in the practice of speech therapy.

Under the auspices of the Federal government, a number of retraining centers were created in military hospitals. These were usually intensive units, manned by a team of clinical psychologists, speech therapists, and various kinds of educators. The results of these war-time rehabilitation experiences naturally led to a demand from the civilian public for the same kinds of services. Since then, a number of civilian centers have been established.

As a direct outgrowth of a lengthened life span, there is an increased incidence of cerebral vascular disease and of patients who have residuals of brain damage from accidents, surgical procedures, and other diseases. While many view this exploding geriatic and chronic disease population as an indictment of modern medicine, others view it as a tribute. The prevailing post-war idea that modern medicine can offer help, to anyone and everyone who needs it, added to the climate which introduced aphasia rehabilitation.

Concomitant with this feeling is the idea that somehow everyone shares the right and the privilege to receive every known therapy for every known disease, provided that funds or insurance coverage are available to cover the cost.

The post-war decade, then, provided a natural climate to stimulate the events which contributed to the concept that aphasic patients could be retrained. In a post-war milieu of inflation, increased social welfare legislation, and improved medical care, the disabled American was, so to speak, brought "out of the closet." A more enlightened public became aware that hiring the handicapped was good business. The word stroke became part of every man's everyday vocabulary. We regularly read accounts in newspapers about famous men like Mencken, Churchill, and Eisenhower who had suffered strokes with aphasic language impairment. Booklets and pamphlets became available to the general public interpreting the problem of aphasia. There was an indication in all of this public information that there were methods of rehabilitating aphasic patients. Better than 250,000 copies, in five languages, of the booklet Understanding Aphasia[1] have been purchased since 1958. This surely reflects a general public's orientation to rehabilitation and an intensive quest for information.

The speech therapy profession was naturally called upon, during the post-war era, to treat the large number of civilian aphasic adults who were referred to speech clinics, university clinics, general hospitals, and rehabilitation centers. University training programs that had no tradition for offering a specialized curriculum in aphasia, added such training. Convention programs of the American Speech and Hearing Association gradually incorporated a number of papers on aphasia. To this day, the recent speech therapy graduate still complains that he is professionally responsible for the treatment of a disability for which he has not been adequately trained. In light of the public's demand for treatment, many types of institutions and professions adjusted their operations to accommodate the population of aphasics demanding treatment. An estimated million and a half persons comprise this population. The community rehabilitation center, the speech therapy departments in general hospitals, city, state, and federal agencies, the resident physicians in training in physical medicine, grants for training, and federal aid to cover courses like the one we share this week, are all post-war phenomena.

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[1] Martha L. Taylor, Understanding Aphasia (New York: The Institute of Physical Medicine and Rehabilitation, 1958).

While those engaged in aphasia rehabilitation continue to conclude that patients do improve with speech therapy, it may well be that our post-war focus on aphasia rehabilitation has far exceeded our knowledge of treatment and our capacity to render effective service. Indeed, a long overdue examination of the treatment of the aphasic patient is in order.

Approaches To Treatment

There are probably as many approaches to aphasia therapy as there are patients. Aphasia therapy is rarely the same thing in any two treatment settings. Indeed, the lack of uniform approach has deterred any carefully controlled studies of the effects of language re-training in the aphasic populations. The methods and rationales described in the literature, and practiced in most settings where aphasic patients are seen, appear to break down into three large general categories. I have labelled these approaches: (1) The Non-Specific Stimulation Approach; (2) The Specific Stimulation Approach; and (3) The Psycholinguistic Approach. In actual practice, these categories frequently overlap. My criteria for classification is based on the belief that if one were to observe actual treatment in many parts of the country, and describe samples of these sessions, one would find a significantly high percentage of time being invested in the practices that are suggested in the label for each category. Some of these follow a specific theoretical model; others are based on results of specific test performance.

A. The Non-Specific Stimulation Approach

The first treatment school, under the category of Non-Specific Stimulation, I have called the Spontaneous Recovery Approach. This approach represents no approach at all. It supports the attitude that aphasic patients cannot recover language except for that recovery which is effected naturally or spontaneously. This school, shared by many physicians, believes that aphasia is an irrevocable state, changeable only through natural processes. Medical history would tend to support this view probably more than any other.

The Environment Stimulation Approach suggests that recovery from aphasia takes place under the combined effects of spontaneous recovery and verbal stimulation. By verbal stimulation it is implied that one's verbal environment should be filled with as much verbalization as possible. One of the sub-groups in this approach holds that the auditory process is the most important communication practice affecting the course of recovery.

This approach is followed by many physicians, and considers the therapeutic management of an aphasic patient to be a process of advising the patient's family and friends to provide a significant quantity and intensity of verbal stimulation during the course of the first year following brain damage. Visitors, reading aloud, television, radio, movies, and conversation are frequently suggested as essential to this treatment. The underlying principle is that since children and foreign speakers acquire language skills through listening and seeing, so does the aphasic adult. This approach reflects the idea that language recovery takes place irrespective of, and incidental to, the kind of stimulation--provided that the stimulation is of a verbal kind. The approach does not consider the kind of verbal stimulation, but simply its quantity and intensity. It does not recognize the linguistic features of the language or a hierarchy of linguistic function.

The Rapport Approach (sometimes called "developing a relationship") is represented by a large number of clinicians. In this approach, the content or method selected for the training process is not considered--provided that a warm relationship between the clinician and the patient is in progress. The patient's gratification as a person is the focus of his language rehabilitation management. This school believes that communication skills, within the limit of the patient's learning potential, will be recovered if a warm interaction has been established. This method is sometimes called a "conversational approach."

The Socialization Approach, which is represented in the major part of the speech pathology literature on this subject, follows the principle that recovery from aphasia is achieved through socialization. This is usually accomplished in group sessions where singing, crafts, hobbies, games, puppets, telling jokes, and even playing pranks are the bases for treatment. The approach has sometimes been cynically referred to as "coffee hour treatment." While this school may utilize a different vehicle from the rapport approach, the goal is essentially the same: motivation via socialization and improved social adjustment through group interaction. The same principles and activities are often practiced in individual treatment.

The Psychotherapeutic Approach usually takes place in group settings and makes little or no direct attempt to retrain language. The focus is on the psychological aspects of aphasic behavior, particularly on problems of anxiety and loss of self-esteem. Clinicians who utilize this approach generally feel that language behavior can be modified in its aphasic characteristics as a secondary gain of psychotherapy.

The Interest Approach attempts to stimulate interest, on the patient's part, in the treatment stimuli by virtue of its subject matter. This usually refers to the patient's known pre-morbid interests. Lexical or vocabulary units, reading, pictures, and other stimuli related to the patient's interests, generate the treatment activity. The stimuli may be selected by the clinician or, in some cases, the patient does the selecting indirectly by showing his interest in a particular activity. Films based on current events and other topics of interest have been suggested in the literature as a possible vehicle for stimulation.[2]

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[2] Joseph Wepman and Anne Morency, "Filmstrips as an Adjunct to Language Therapy for Aphasia," Journal of Speech and Hearing Disorders, XXVIII (May, 1963), 191-194.

B. The Specific Stimulation Approach

The *Association Approach* attempts to elicit associated lexical items from the patient by presenting him with stimuli that might recall other words of the same class or of similar semantic value. The purpose of such a session might be to simply recall these words, or the therapist may more specifically attempt to structure the session around so-called "families" of words, according to their referents. The vocabulary is divided into semantic units--such as *body parts*, *house furnishings*, and *foods*. Any modality or context may be used in this approach, provided that the vocabulary remains in the same referent "family." An example of a variation of this method is to present orally, for the patient's repetition, the word *coffee* followed by *a coffee pot*, *hot coffee*, *a cup of coffee*, *the coffee is boiling*, *drink coffee*, and *please pass the coffee*. According to this school, the introduction of an infinite number of possible word environments for the word *coffee* will strengthen the patient's association for the word, hence increase his ability to recall it.

In the *Situational Approach*, everyday situations are acted out. The context of function will hopefully elicit, or facilitate the learning of, the vocabulary and/or the statements that are functionally useful for the particular situation. This approach can specifically teach a script of responses in dialogue fashion or, non-specifically, simply bring forth oral and gestured expression in the context of a common situation. The premise for this approach is based on the clinical observation that, in everyday situations, aphasics often produce responses which they are incapable of producing under task-oriented conditions.

Another specific stimulation approach is represented by those who use *Auditory Stimulation* as the foundation for all aphasia treatment. This group believes that patients improve not only in their ability to understand, but in their ability to read and write, when auditory stimulation is employed. This approach is more method oriented than most of the others mentioned. The length of each unit and word frequency comprise the basis for the choice of materials and measurement of progress. Data have been presented in the literature which support this concept.

The Minimal Differences approach is based on the idea that one of the aphasic patient's primary difficulties is the kinesthetic and/or visual recognition of similar small language units: the written letters and phonemes. Minimal word pairs, similar sounding words, and minimal orthographic differences in words are often used as stimuli for teaching.

A general over-all view of the commonly practiced approaches to aphasia therapy certainly reveals a willingness, indeed an eagerness, on the part of the aphasia clinician to help aphasic patients. An enormous amount of tender loving care, understanding, and kindness have been invested in caring for aphasic patients. Some common sense, in the absence of a body of specific knowledge in aphasia rehabilitation, has certainly been applied. But, for the most part, the short history of aphasia therapy is dominated by random trial and error methods, based on improvisation and sometimes "intuition." It seems most workers believe that aphasia rehabilitation is a stimulation process rather than an educative one, despite evidence--however limited--to the contrary. Techniques of many kinds have been applied with little or no knowledge of their effectiveness or expectation. Wepman[3] has said: "All varieties of language disturbances seem to get some resolution of their problems, seemingly regardless of the particular approach used. . . ." Perhaps we should say that aphasic patients have improved in spite of, rather than because of, the approach used.

One of the greatest limiting factors in the progress of the history of aphasia rehabilitation is the fact that the speech therapist has been relegated to do the job. This is a young field and we could hardly expect that the principles and methods inherent in articulatory improvement, for example, or cleft palate therapy, could have application to recovery from a verbal impairment as complex and broad as aphasia.

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[3] Joseph M. Wepman, "A Conceptual Model for the Processes Involved in the Recovery from Aphasia," Journal of Speech and Hearing Disorders, XVIII (March, 1963), 5.

C. The Psycholinguistic Approach

The least common approach to the rehabilitation of aphasia I shall call the Psycholinguistic Approach. There is little, if any, literature which supports or describes this approach. Scargill[4] is perhaps the only writer in speech pathology literature who directed his attention to the application of contemporary linguistic theory to recovery from aphasia. What I am about to describe, then, represents an approach used by a very limited number of clinicians.

No one would deny that motivation, fatigue, premorbid and present personality, anxiety, and other factors must be considered in the total management of any aphasic patient. Nor can it be said that these factors do not affect a patient's recovery. But the primary objective of aphasic rehabilitation is language learning. Hence, the task of the aphasia therapist is a teaching task. A theoretical framework for language learning can be derived from psycholinguistics.

Psycholinguistics is a term which describes the combined fields of behavioral psychology (the science of behavior) and linguistics (the science of language). Those who follow a psycholinguistic approach to the therapy of aphasia believe that (1) a sound behavioral psychology can provide the teaching methods which will facilitate language learning and retention, and that (2) a sound linguistic basis can provide the systematic analysis and description to guide the content and order of presentation of the linguistic elements we wish to teach. The application of psycholinguistic theory to the treatment of aphasia offers the possibility that there is a science of aphasia rehabilitation.

Linguistics is not a fad, but a growing body of knowledge and theory. The field has changed markedly during the past fifty years. The traditional linguist (sometimes called the "prescriptive grammarian") is indeed a thing of the past. The prescriptivist believed that preset rules should determine the way in which language is used. He concerned himself with prescribing the correctness of grammar and vocabulary. Many of us are familiar with English teachers who teach grammar by traditional methods. The traditional linguist fosters the notion that language usage must be governed by pre-established rules of correctness and that language must be preserved and not permitted to change. The traditional linguist often analyzes language according to its written form rather than its spoken form. The spoken language certainly does not adhere to the traditional linguist's notion of correctness. In English, in particular, the spoken and written languages are vastly different.

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[4] M. H. Scargill, "Modern Linguistics and Recovery from Aphasia," Journal of Speech and Hearing Disorders, XIX (December, 1954), 507-514.

Modern-day linguistics is called "descriptive linguistics." The descriptive linguist believes that language is primarily an oral-aural event. He is concerned with the science of describing what the speakers of a language say. He believes that the "rules" of a language are determined by what its speakers say, not what someone thinks they ought to say. Today's linguistic scientist constantly strives for refined methods of describing the oral event. He describes what is said, according to linguistic systems of description: phonology, morphology, syntax, and semantics. Each of these broad descriptive categories can be reduced to narrow sub-systems of language description.

The most rapidly growing area of linguistics is called "structural linguistics." This term refers to a kind of linguistics which is primarily interested in discovering and describing, as concisely and accurately as possible, the interrelationships and patterns which make up the structures of languages. In a way, structural linguistics can be called the mathematics of language. It is the most rigorously scientific form of linguistics.

To demonstrate the linguistic significance of structures, and function words that operate as the skeleton for the structures, linguists often cite the "Jabberwocky" from Alice in Wonderland. In this poem, the function words manage to hold together groups of nonsense words and convey meaning through structure. If nonsense words were substituted for the function words, the meaning conveyed by the structure would not be transmitted. One can easily observe this by examining the first two lines: "Twas brillig and the slithy toves did gyre and gimble in the wave."

Syntax is the body of rules by which words are put together to form sentences. The linguist has clarified two aspects of language: (1) the rules, structures, and transformations that make up the syntax of the language; and (2) the classes of items or parts of speech that the syntax orders. The classes of words are defined as groups of words having similar "privileges of occurrence"; that is they can appear in the same context. The structural linguist, in his laborious task of analyzing what is said, tells us that oral language consists of specific mathematically determined structures. The grammar generated by these structures is predictive, in that many other sets of words can be assigned the same order of occurrence in these structures. Children acquire all of the structural grammar necessary for language usage at a very early age; probably before they are seven years old. The remaining years of language acquisition are devoted to the mastery of vocabulary.

Children often demonstrate their skill in manipulating structures before vocabulary when they produce a sentence and substitute a nonsense word for a vocabulary item they do not know. The child invariably placed the unknown word in its proper position in the sentence. Children seem to appreciate grammatical function quite in advance of mastering a large vocabulary.

The linguist recognizes that the structures of the language can be viewed and analyzed systematically and mathematically, whereas the meanings of words cannot. The unlimited number of variable meanings attached to words cannot be systematically sorted out.

The linguist views language as a system of sounds, uttered so habitually that we are not conscious of the movements or the patterns in which these sounds appear. The learning of language, in the linguist's view, is not a magical and mysterious process without underlying rationale. It is primarily dependent upon the pattern practice of the fundamental structures of the language. These sentence patterns have been identified and analyzed and follow a logical sequence, according to a syntactic order of difficulty.

In order for a language to be "learned," an individual must have automatic control of the structures of the language. This is accomplished through the imitation and repetition of the patterns of the language. The process of the formation of language behavior is divided into five general, yet distinguishable, stages of learning:

First stage: recognition.

Second stage: imitation; echoing the model performance.

Third stage: repetition of model based on memory of echoed performance.

Fourth stage: variation of third stage.

Fifth stage: spontaneous selection of a response from a repertoire of learned responses.

To facilitate the establishment of language habits, the linguist suggests that in the process of pattern practice (1) utterances must be short. Patterns should be built only on those structures and vocabulary already learned. (2) Vocabulary should be selected on the basis of frequency of occurrence and only one item at a time should be taught. (3) The immediate reinforcement of a correct response is imperative. (4) Different linguistic features should not be mixed until the response of the individual features of the material to be taught has become automatic.

This brief overview of certain linguistic principles is directly relevant to the language rehabilitation of the aphasic patient. A review of the most common practices in the treatment of the aphasic would reveal that the greatest emphasis, linguistically speaking, has been on teaching aphasic patients vocabulary. Little, if any, attention has been given to the orderly teaching of the basic structural patterns of the language which might accommodate the vocabulary. When "grammar" is handled by the speech clinician, it is usually along traditional prescriptive lines. The linguist has demonstrated that the semantic aspects of the language are the least classifiable features of the language. Yet, most aphasia therapy has focused on the "meanings" of words reflecting, as pointed out above, a random and illogical practice.

There have been some linguistically based studies in aphasia which confirm some linguistic principles. The work of Dr. Harold Goodglass has very particular relevance to aphasia treatment. One of his findings is that the plural forms of words are more likely to be retained by aphasic patients than the possessives, despite the fact that these may be identical in their phonemic content, for example, the word bills (meaning the plural of dollar bills) and the possessive Bill's book. While these two words are phonemically identical, the operation they perform is different and hence lends each a different place in the hierarchy of difficulty. The implication of this finding to treatment is obvious. Studies of this sort with aphasic patients are few.

Let us now examine some behavioral psychology principles, with special reference to a relatively new area called "programmed learning." This is not to be confused in any way with teaching machine devices. Programmed learning is really nothing more than a system for finding out something about learning. The field reflects the principle that the conditions for learning can be so arranged that an individual's responses are under the control of the program. In applying this to aphasia treatment, the implication is that the control of the patient's response is in the hands of the therapist and that by properly arranging stimuli, a series of successful responses can be assured, reinforced, and hopefully incorporated in the patient's repertoire of responses. The following characteristics differentiate programmed instruction from other types of teaching. (1) A set of specifications of the instructional goals (terminal behaviors) is detailed. (2) The material of instruction is organized into such a carefully designed sequence of small steps that each step is made easier, by virtue of the material previously mastered. In practice, it is found that the optimal step-size is usually much smaller than one might think. (3) The learner must be properly reinforced by confirmation of correct responses. In aphasia therapy, most reinforcement has consisted simply of praising the patient. We recently observed an example of an over-reinforced aphasic patient, who ended all utterances with "Good, that's fine." The reinforcement technique of praise had obviously been misused. It no longer operated as reinforcement, but had become an integral part of the patient's verbal behavior. (4) The learner must actively participate in the learning process. In order for responses to come under the control of the learner, he must actively take part in their production.

Programmed learning can be self-instructional, or it can be presented by a teacher or a machine. The rationales for programmed instruction refer in part to "operant conditioning." Any response that can be rewarded, and which as a result tends to occur more frequently, is an operant. Most of the responses we are interested in teaching aphasics are operants. If we desire to change behavior, one way to do it is to wait for the desired response to occur and then reinforce it. But an instructor who follows principles of programmed instruction believes that behavior can be shaped. The term "controlling behavior" can be misunderstood. This does not describe a puppeteer, but refers to the fact that the behavioral scientist believes matters can be arranged in such a way that new responses will come under the control of specific stimuli.

All of this can be generally summarized this way: decide what responses you want to teach; arrange matters so that these responses occur as frequently as possible--with emphasis on success rather than error; reinforce the successful responses.

A few months ago, we began to consider the possibilities of using some programmed instruction with global and other severely impaired aphasic patients that had not responded to the "conventional" methods of aphasia rehabilitation. The patients selected for experimentation were those who were unable to perform any of the items on our language tests. The subjects had over-all scores on the Functional Communication Profile of less than 10%.

Our first task was to specify the terminal behaviors we wished to teach. Since the subjects for whom we would design programs were nonverbal, we decided that the first programs should attempt to teach those processes we considered prerequisite to verbal learning. This meant that those modalities associated with verbal skills would be omitted from the initial programs. There would be no terminal behaviors in the areas of oral production, reading skills, writing skills, or auditory comprehension. The resulting Pre-Verbal Programs were designed according to the following scheme:

PRE-VERBAL PROGRAMS

Activity		Modality	Terminal Behavior
		I. IMITATION	
Program 1.	Imitation of body movements.	Movement	Ability to imitate gross body movements.
Program 2.	Imitation of fine oral movements, gross phonation and articulation.	Movement	Ability to imitate oral movements, produce gross pitch variations, imitate vowels, imitate limited number of consonants.
		II. VISUAL RECOGNITION	
Program 1.	Matching and selecting.	Content: pen, book; 1, 2; red, blue; objects; blocks; flashcards, with and without size variation.	Ability to match and select like and unlike stimuli.
Program 2.	Matching and selecting.	Same as above with size variation and addition of printed flashcards.	Ability to match and select material with printed and written words.
		III. PRE-WRITING	
Program 1.	Tracing and copying.	Circles, squares, triangles, horizontal and vertical lines.	Tracing and copying of geometric forms.
Program 2.	Fading and filling in.	Same content as Program 1.	Completion of geometric forms with reduced visual cues.
Program 3.	Tracing, copying, fading.	Writing name.	Ability to trace, copy and fill in last name. Ability to write last name spontaneously.

Each terminal behavior was analyzed and broken down into small steps. A mathematical formula for the teaching sequence was the result of this analysis. In the presentation of all of the programs in the pre-verbal series, no oral-aural interaction between the clinician and the patient was permitted during the administration of the program. The Holland-Skinner Constructed Response type of program in a linear arrangement was chosen as a format for stimuli presentation.

A sample page, from Program 1 in the "Visual Recognition" sequence, is presented on pages 156-159, to give some idea of the step-size and specification of stimuli. This particular program includes more than 230 steps, designed to reach the terminal behavior of matching and selecting objects and picture units with two color and two size variations.

In order to reduce the many possible variables that might be inadvertently introduced during administration of the program, all of the stimuli selected for teaching was meticulously designed and made for the specific programs. For example, in the case of the red and blue blocks used to teach color matching, three different sizes of blocks were used and the program script indicated precisely at which point in the teaching sequence the specific size variations were to be introduced. The clinicians were instructed that the program script must be rigorously adhered to, not only in the stimuli presented but in the exact order of presentation. Data werecollected for each administration of a program. These data provided the feedback necessary to improve the programs and to analyze their effectiveness.

The choice of the object, picture, and flashcard stimuli that were to be used as vehicles for nonverbal teaching, was based on certain linguistically determined criteria. The criteria for choice of the six lexical units--a pen, a book, blue, red, 1, and 2--were: (1) ease of phonemic elements; (2) monosyllabic words; (3) size of objects permitted ease of presentation on a table top; (4) regularity of article required by noun; (5) regularity of grammatical operation; (6) functionally useful vocabulary; (7) ease of combining these later into structures without grammatical compromises; (8) ease of visualization of item; (9) facilitation for teaching singular and plural usage in structures later; and (10) high frequency of occurrence in spoken language.

Naturally, no one of these vocabulary items met all of the pre-determined criteria for selection. Nevertheless, let me give an example of a commonly selected vocabulary item which meets very few of the criteria mentioned. The word apple carries the irregular article an; is bisyllabic; does not have a particularly high probability of occurrence in the language; has a silent final letter; has limited functional usage (except for those particularly fond of apples!); and is phonemically complex.

Our over-all plan was to carry the patient through these pre-verbal programs, then on to programs designed to teach the same lexical items in step-by-step arrangements in each modality--reading, writing, auditory comprehension, and oral production. Depending on the type of program, the stimuli may be presented by a clinician or on a variety of teaching machines. The total number of steps involved in the complete teaching program on this level is better than 5,000. The actual number of teaching sequences (number of individual programs) is about 20. In all cases, terminal behaviors for each program are carefully specified; review sequences are introduced at certain points, to "test" the efficacy of learning; and the cues provided in each subsequent program are gradually reduced.

The results obtained in this pilot project have been extremely exciting. They have surpassed our most optimistic expectations. Patients who were unable to learn through other teaching techniques have not only mastered all of the pre-verbal skills, but have proceeded to acquire skills on a much higher level in auditory comprehension and oral production. In one of these cases, the patient's verbal impairment was of over one year's duration and considered to be irrevocable.

Certain observations in this particular informal pilot program are of special interest. While the material appears monotonous and repetitious, patients have demonstrated maximum attention to the presentation of programmed materials. They have evidenced a persevering capacity with this method, whereas other methods usually did not hold their attention. The anxiety, so blatantly expressed in many severely impaired aphasic patients, was reduced dramatically. Patients who had numerous catastrophic reactions to other methods, did not exhibit any catastrophic symptoms. Patients with minimal fatigue thresholds, tolerated much longer periods of training.

In addition, our experience in programming instruction for aphasic patients in this pilot project has confirmed many of the advantages often cited. Programmed instruction: (1) enables patient to work at his own rate of language learning; (2) has built-in measurements of language learning; (3) forces the clinician to work at patient's real level of functioning; (4) has built-in systematic record of responses to act as feedback for programming; (5) forces us to analyze terminal behaviors and approximately design materials and methods for their realization; and (6) forces and facilitates a markedly increased precision in the description of language recovery for charts, progress notes, research, and so forth.

I have only told you of the positive and rewarding aspects of this technique in aphasia rehabilitation. But there are some significant disadvantages. The greatest, perhaps, is that therapists somehow do not like programmed instruction. They continue to believe that the therapist's role in aphasia rehabilitation should be creative. It should also be noted that the amount of careful planning, analysis, and expense required for the design of the simplest program is extraordinary.

B. F. Skinner[5] has made certain points about the challenge of programmed instruction clear. He says:

> The traditional teacher may view these programs with concern. He may be particularly alarmed by the effort to maximize success and minimize failure. He has found that students do not pay attention unless they are worried about the consequences of their work. The customary procedure has been to maintain the necessary anxiety by inducing errors.
>
> Difficult as programming is, it has its compensations. It is a salutary thing to try to guarantee a right response at every step in the presentation of a subject matter. The programmer will usually find that he has been accustomed to leave much to the student--that he has

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[5] B. F. Skinner, "Teaching Machines," Science, CXXVIII (October 24, 1958), 975.

> frequently omitted essential steps and neglected to invoke relevant points. The responses made to his material may reveal surprising ambiguities. Unless he is lucky, he may find that he still has something to learn about his subject. He will almost certainly find that he needs to learn a great deal more about the behavioral changes he is trying to induce in the student. This effect of the machine in confronting the programmer with the full scope of his task may in itself produce a considerable improvement in education.

We should be encouraged by all the recent experiences in treating aphasic patients which have come from the area of psycholinguistics. We are apparently on the brink of creating a science of aphasia rehabilitation. Our increased effectiveness should ultimately provide the most efficient and maximum verbal recovery within the limits of each aphasic patient's disability.

VISUAL RECOGNITION[a]

(Program 1)

DIRECTIONS A (DO NOT TALK)

Stimuli

1. Place one object before patient. Gesture to patient that he is to examine the object.
2. Produce matching object from hidden place (drawer, pocket, etc.). Place second object next to first object.
3. Remove objects and give one to patient.
4. Bring second object toward the first, indicating through gesture that patient is to do the same thing.
5. Continue repeating steps 1-4 until patient responds by bringing the two objects together.

Response

1. Place both objects before patient at least one foot from each other.
2. Patient brings both objects close together.

Reinforcement

1. Regardless of patient's performance: perform steps 2, 3, and 4 of stimuli procedures.
2. If patient's response has been correct, indicate this non-verbally by smile, head nod, etc.

DIRECTIONS B (DO NOT TALK)

Stimuli

1. Place the three objects before patient.
2. Pick up the two like items.
3. Put them down again.
4. take the patient's hand and help him to pick up the same like items.
5. Repeat the first three steps.

Response

1. Gesture to patient that this time he is to match, just as he did in preceding step.
2. Patient selects the two like items.

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Reinforcement

1. Regardless of patient's performance: perform steps 2 and 3 of stimuli procedures.
2. If patient's response has been correct, indicate this non-verbally by smile, head nod, etc.

DO NOT INTRODUCE NEW STIMULI UNTIL PATIENT HAS SUCCESSFULLY PERFORMED ALL OF THE ABOVE STEPS!

Directions A

1. Matching Identical Objects

one pen and one pen
one book and one book
one pen and one pen
one book and one book
one pen and one pen
one pen and one pen
one pen and one pen
one book and one book
one book and one book
one book and one book

Directions A

2. Matching Objects with Variation in Size

one pen (large) and one pen (small)
one pen (small) and one pen (large)
one book (large) and one book (small)
one book (small) and one book (large)

Directions B

3. Selection of Two Identical Objects

one pen and one pen and one book
one pen and one pen and one book
one pen and one pen and one book
one book and one book and one pen
one book and one book and one pen
one book and one book and one pen
one book and one pen and one pen
one book and one pen and one pen
one book and one pen and one pen
one pen and one book and one book
one pen and one book and one book
one pen and one book and one book

Directions B

21. Selecting Two Like Colored Pictures

one blue pen picture and one blue pen picture and one red pen picture
one red pen picture and one red pen picture and one blue pen picture
one blue book picture and one blue book picture and one red book picture
one red book picture and one red book picture and one blue book picture

Directions A

22. Matching 2 Like Colored Objects and Pictures

one blue pen object and one blue pen picture
one blue pen picture and one blue pen object
one blue book object and one blue book picture
one blue book picture and one blue book object
one red pen object and one red pen picture
one red pen picture and one red pen object
one red book object and one red book picture
one red book picture and one red book object

Directions B

23. Selecting Two Like Colored Objects

one blue pen object and one blue pen object and one blue pen picture
one blue pen picture and one blue pen object and one blue pen object
one blue book object and one blue book object and one blue book picture
one blue book picture and one blue book object and one blue book object
one red pen object and one red pen object and one red pen picture
one red pen picture and one red pen object and one red pen object
one red book object and one red book object and one red book picture
one red book picture and one red book object and one red book object

Directions A and B

24. REVIEW

one book object and one book picture
one book picture and one book picture and one book object
one red pen object and one red pen object
one red book object and one red book object and one red pen object
one blue pen picture and one blue pen picture
one red pen picture and one red pen picture and one blue pen picture

Directions A

25. Matching Like Color Object (with Variation in Size) and Picture

one blue pen object (small) and one blue pen picture
one blue pen picture and one blue pen object (small)
one blue book object (small) and one blue book picture
one blue book picture and one blue book object (small)
one red pen object (small) and one red pen picture
one red pen picture and one red pen object (small)
one red book object (small) and one red book picture
one red book picture and one red book object (small)

Directions A

26. Matching Like Color Object and Picture (with Variation in Size)

one blue pen object and one blue pen picture (small)
one blue pen picture (small) and one blue pen object
one blue book object and one blue book picture (small)
one blue book picture (small) and one blue book object
one red pen object and one red pen picture (small)
one red pen picture (small) and one red pen object
one red book object and one red book picture (small)
one red book picture (small) and one red book object

Directions A

27. Matching Objects and Colored Blocks

one blue pen object and one blue color block
one blue color block and one blue pen object
one blue book object and one blue color block
one blue color block and one blue book object
one red pen object and one red color block
one red color block and one red pen object
one red book object and one red color block
one red color block and one red book object

LINGUISTIC CONSIDERATIONS IN LANGUAGE THERAPY

Ralph Stoudt

Linguistics is a science which deals with language. The utterances of oral communication are the raw materials with which the linguist works. The sound system and the grammar of a given language are focal points for the linguist. His purpose is to discover how a language works. His goal is to formulate a set of rules which will permit the generation of all possible correct utterances in that language.

Speech pathology also deals with language. The utterances of oral communication are the raw materials with which the speech pathologist works. His purpose is to modify disturbed language patterns in the direction of more normal patterns. His goal is to facilitate effective oral communication. In these activities the language therapist cannot do any other than work with sound systems and grammar. Such endeavor demands understanding of normal language behavior.

At this point the interests of the linguists and the speech pathologists converge. Both disciplines have made significant strides in their study of language. Until recently however members of each group have tended to pursue their respective interests with little sharing of information. This lack of communication was probably not due to an unwillingness to share but rather to an unawareness on the part of each party of the depth and scope of the other's understanding of the problem at hand.

Roman Jakobson, an eminent linguist, is a pioneer in bringing about cooperation between linguists and others interested in language disturbance. Applying the strictures of purely linguistic criteria to the interpretation and classification of aphasic facts, Jakobson has developed two major hypotheses concerning the nature of aphasia. These theories reflect the linguist's two major concerns in the study of language: grammar and the sound system.

Most familiar, perhaps, is Jakobson's theoretical explanation of aphasic phenomena at the level of grammar. He discusses the disturbance in grammar in terms of similarity disorder and contiguity disorder. Similarity disorder is roughly equivalent to what has been described in more traditional terminology as word finding difficulty. Contiguity disorder has been described primarily as a difficulty in using the syntax of a language. These interpretations of language disturbance at the grammatical level have been widely acclaimed and accepted.

The second hypothesis, less well known and certainly not as widely accepted, concerns the sound system of the language. This phonemic regression hypothesis suggests that the phonemic production of aphasic patients is a mirror image of the development of sounds in children's language.

"Phonemic regression" seems to be a logical extension of some aspects of the contiguity disorder referred to a moment ago. Perhaps Jakobson should speak for himself on this point.

> The comparative description of the phonemic systems of diverse languages and their confrontation with the order of phonemic acquisitions by infants learning to speak, as well as the gradual dismantling of language and of its phonemic pattern in aphasia, gives us important insight into the interrelation and classification of the distinctive features. The linguistic, especially the phonemic progress of the child and the regression of the aphasic obey the same laws of implication. If the child's acquisition of distinction B implies his acquisition of distinction A, the loss of A in aphasia implies the absence of B, and the rehabilitation of the aphasic follows the same order as the child's phonemic development.[1]

Jakobson contends that the development of sounds in children's language shows a time order of great regularity.

The implications of the phonemic regression hypothesis for language retraining are easily apparent. If such a regular sequence for development of sounds is real, then any program of language retraining which has to deal with the sounds of the language will have to take this regular system into account at the planning stage. Establishment of definite priorities in sound training would be most helpful in dealing with many aphasic patients. Nor should we neglect the corollary of this proposition. The regularity of such a scheme would provide a tangible means of determining progress in the therapy program.

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[1] R. Jakobson and M. Halle, Fundamentals of Language ('S-Gravenhage: Mouton and Co., 1956), pp. 26-27.

These two considerations, planning procedures and measuring progress, hold a central place in any program of language retraining. Those who work daily in language therapy cannot afford to overlook any possible guidance. It is appropriate then to review the phonemic regression hypothesis in greater detail.

The concept of "phonemic regression" is surely not unique with Jakobson. Scattered throughout the literature there are premonitions of the idea.

Preyer envisaged the main points of this concept in 1889 when he wrote:

> The normal child that does not yet speak perfectly resembles the diseased adult, who for any reason, no longer has command of language. And to compare these with each other is the more important, as at present, no other way is open to us for investigating the nature of the process of learning to speak: but this way conducts us fortunately, through pathology to solid important physiological conclusions.[2]

Henry Head pointed out that the speech of aphasics "sometimes closely resembles baby language."[3]

In his work, Infant Speech, Lewis suggested in 1939: "It is of very great interest to notice that the defects which children's speech shows . . . are very similar to those which occur in the speech of aphasics."[4]

Also, in 1939, Alajcunine concluded:

> It is our impression that there are strong analogies between pathological phonetic alterations and the first manifestations of infantile language. . . . Numerous changes which can be observed during different stages of recovery in our patients are also found in the normal phonetic progress of the child.[5]

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[2] W. Preyer, The Mind of the Child, Part II: The Development of the Intellect, trans. H. W. Brown (New York: D. Appleton and Co., 1898), p. 34.

[3] H. Head, Aphasia and Kindred Disorders of Speech, 2 vols. (London: Cambridge University Press, 1926), I, 231.

[4] M. Lewis, Infant Speech: A Study of the Beginnings of Language (New York: Harcourt, Brace and Co., 1936), p. 186.

[5] T. Alajouanine, "Verbal Realization in Aphasia," Brain, LXXIX (March, 1956), 21.

It is out of this historical background that Jakobson writes clearly and directly about "phonemic regression," in this way:

> There is one level of aphasic phenomena where amazing agreement has been achieved during the last twenty years between those psychiatrists and linguists who have tackled these problems, namely, the disintegration of the sound pattern. This dissolution exhibits a time order of great regularity. Aphasic regression has proved to be a mirror of the child's acquisition of speech sounds, it shows the child's development in reverse.[6]

Until Jakobson's formulation of the phonemic regression hypothesis, the use of children's speech as a model for aphasic language disturbance was largely illustrative. Jakobson's approach attempts to construct a theoretical explanation of language disintegration which incorporates phonemic regression as a fundamental tenet. The theories of Jakobson concerning language breakdown attempt to relate the disintegration of language to normal language function. In order to understand Jakobson's theories concerning the breakdown of language function, it is imperative to review his concept of language in general.

Language for Jakobson consists of two basic processes: selection of linguistic units and combination into higher orders of complexity. These two processes, selection and combination, summarize all language activity. A speaker selects sounds (phonemes) from the store of those used by his language, and combines them into words. Morphemes (minimum units of meaning) may be selected and combined with the phonemes in the formation of words. These selected words are combined into sentences. Utterances are combinations of sentences.

Hence any meaningful or lawful grouping of linguistic signs binds them into a superior unit. This means that any linguistic unit, at one and the same time, serves as context for simpler units and/or finds its own position in the context of a more complex linguistic unit. Linguistic signs or units are arranged in an ascending hierarchy: phoneme, morpheme, word, sentence, and utterance.

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[6] Jakobson and Halle, op. cit., p. 57.

Disordered communication in aphasia, implies a disruption of these basic language processes. Jakobson suggests that two major types of language disintegration in aphasia are related to the two processes of language we have just described. The first type of language disturbance, similarity disorder, implies a diminution of the selective capacity of the speaker. Generally similarity disorder is seen in the subject who has difficulty in using specific nouns in supplying exact or near synonyms. The second type of disorder, contiguity disorder, is related to the speakers ability to combine linguistic units into larger wholes. The aphasic with contiguity disorder tends to the use of one sentence utterances, of a very infantile nature, or to one word sentences.

Jakobson maintains that continguity disorder is not limited to the utterance and sentence levels in the hierarchy of linguistic units. You will recall that a linguistic unit is both a constituent part of a superimposed context, and is itself a context imposed upon smaller constituents. Thus a word may be a part of a sentence context, and itself be a context with phonemes and morphemes as constituents. Jakobson sees the effects of contiguity disorder at the word level and at lower levels in the hierarchy of linguistic units.

Contiguity disorder is evident at the word level, and below, in several ways. Frequently inflection is lost. The patient tends to use the infinitive for finite verb forms. He is unable to separate words into their roots and derivational suffixes. In those languages with declension, the nominative case is substituted for all the others. Often the patient is unable to resolve a compound word into its components.

Finally, the aphasic tends to abolish the hierarchy of linguistic units. The scale of these units is collapsed to a single level. This final level consists of either a class of significative values, the word, or a class of distinctive values, the phoneme. In cases where significative values persist, the word becomes the smallest discriminable unit. If the class of distinctive values retains dominance, then the phoneme is the smallest discriminable unit. The patient is able to distinguish, identify, and reproduce phonemes.

When the word becomes the smallest discriminable unit, the patient is unable to resolve the word into its constituent phonemes. To use Jakobson's examples, he is able to recognize and discriminate common words such as pig, dig and big. He is quite deficient, however, in his ability to discriminate unfamiliar words containing different orders of the same phonemes such as gib, gip, and gid. He is also unable to discriminate nonsense syllables.

Jakobson suggests that when the normal speaker of English is confronted with such syllables composed of English phonemes in an acceptable order, he is able to make three generalizations about them. First, they consist of standard English phonemes. Second, since the rules of phonemic arrangement in English are not violated, they are recognized as possible words in English. Third, if these syllables are in fact English words, they undoubtedly refer to different things because of the sound contrasts. The aphasic, unable to make these phonemic distinctions cannot make these generalizations. Neither does he recognize them as words. They are meaningless and indiscriminable to him. In this way, a conflict between distinctive and significative functions of the linguistic scale develops.

This conflict between the significative and distinctive functions of the linguistic scale is usually resolved by reducing the inventory of phonemes. The phonemic production of the individual shows a gradual regression.

This is the "phonemic regression" which mirrors the speech development of the child. In this regression the language of the aphasic is reduced by degrees to one sentence, one word, even one phoneme utterances. The patient returns to the initial phases of infantile linguistic development or even to prelinguistic stages.

In this analysis, it has become apparent that phonemic regression in the language of the aphasic is directly related to his discrimination ability. Luria's investigations lend some support to this aspect of Jakobson's hypothesis. According to Luria, disruption of auditory perception is a fundamental and persistent symptom of lesions of the temporal lobe. These lesions do not produce a hearing loss in any part of the frequency range but "inevitably lead to damage in the process of differentiation and generalization of sounds, in other words, the process of sound analysis and synthesis."[7] Subsequent tests of gross discrimination ability indicated that temporal lobe damage seemed to cause difficulty for Luria's patients in making precise phonemic distinctions.[8]

The phonemic regression hypothesis is not universally accepted. Jakobson's assumptions have been attacked on several points. Critchley[9] and Fry[10] have emphasized the great variations in phonemic usage by aphasic patients, in arguing against the regularity of phonemic change. Albright[11] stresses the social and psychological differences between the aphasic and the child. Goldstein sees some value in the study of childhood language for developing insight into the course of language disintegration. He warns strongly against the overzealous use of apparent similarities between the two sequences with this statement: "It should never be forgotten that language is embedded in the total personality and that the distinctions between the personality of children and that of adults may produce essential differences."[12]

The linguistic contributions of Jakobson to the understanding of aphasia were discussed at length at the Boston Research Seminar on Aphasia in 1958. The participants felt that the evidence at hand did not impressively support the phonemic regression hypothesis. The conference concluded that substantiation of the phonemic regression hypothesis required first, that a regular order of phonemic development be observed in children speaking a given language and second, that dissolution in aphasics be inversely related to this regular order.[13]

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[7] A. Luria, "Brain Disorders and Language Analysis," Language and Speech, I (January-March, 1958), 57.

[8] Ibid., p. 18.

[9] M. Critchley, "Articulatory Defects in Aphasia," Journal of Laryngology and Otology, LXVI (January, 1952), 11.

[10] D. Fry, "Phonemic Substitutions in an Aphasic Patient," Language and Speech, II (January-March, 1959), 61.

[11] R. Albright, "Differences Between the Child and the Aphasic," Language and Speech, I (July-September, 1958), 178.

[12] K. Goldstein, Language and Language Disturbances (New York: Grune and Stratton, 1948), p. 35.

Let us examine each of these requirements. Jakobson proposes a theory concerning the development of the sound structure in child language. Briefly, he contends that child language begins (and aphasic dissolution ends) with combinations of polar configurations of the vocal tract. An example is seen in /pa/ which represents the extreme of labial closure opposed to maximum opening of the vocalic tract. In the very early stages of child language this choice between /pa/ and /a/ is the chief carrier of meaning. The next stage in development is the appearance of nasal consonants. The contrast here is between the completely closed oral consonant and the closed main tract with an open subsidiary tract. Jakobson says that "the opposition of nasal and oral consonant, which belongs to the earliest acquisitions of the child, is ordinarily the most resistant consonantal opposition in aphasia and it occurs in all the languages of the world except for some American Indian languages."[14] The process continues with the development of further oppositions and contrasts such as dental versus labial, velopalatal versus labial and dental, and so forth. An analogous series of contrasts is responsible for the evolution of vowels. These sequences of oppositions and contrasts are held to display a time order of great regularity. It would seem that the linguistic environment of the child selects and reinforces the oppositions and contrasts required by the language he is learning. The next logical step, then, is to compare the actual development pattern of children learning language with the model proposed by Jakobson.

Evidence concerning the order of language development is difficult to evaluate. Much of the material bearing on this topic consists of informal accounts of the development of a single child. Differences in methodology, such as type of symbol system used to record utterance, ages at which samples were recorded, and the like make the objective evaluation of most of this material extremely difficult. Twenty years ago, Irwin and Chen[15] commented that a "systematic knowledge of speech sound status throughout the first year of life has not yet been achieved." The extensive work done by Irwin and Chen and others, in the interim, does not seem to have changed the status of our knowledge greatly.

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[13] C. E. Osgood and M. S. Miron (eds.), Approaches to the Study of Aphasia (Urbana: University of Illinois Press, 1963), pp. 68-69.

[14] Jakobson and Halle, op. cit., p. 38.

The problems inherent in child language learning studies make it difficult to determine the order, amount, or rate of dissolution which theory might prescribe for aphasics. Hence a final evaluation of this aspect of the aphasic's problem must await further work in child language development studies.

Let us turn to the second condition which must be met: dissolution of phonemic usage. Although we have seen that it is difficult to relate dissolution to child language development data, the notion of dissolution itself has not been challenged. "Phonemic regression" as it has been considered so far has been seen largely as a productive phenomenon. The question must be raised, at this point, if other factors might contribute to the appearance of a "phonemic regression" from a production standpoint. Dysarthria or apraxia could disrupt the phonemic production of the patient. Such disturbance may or may not follow a regular pattern.

Earlier in this discussion, the difficulty that aphasics experience in making auditory discriminations was presented as one aspect of the dissolution of speech function. Such difficulty in making accurate auditory discriminations was considered fundamental to "phonemic regression."

Auditory discrimination ability is a skill which shows a consistent increase with age, decelerating at about five years of age. This would indicate that auditory discrimination is a skill which develops and becomes stable relatively early in life. Since language development involves a sharpening of discrimination, breakdown in language should show this process in reverse.

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[15] O. Irwin and H. Chen, "Speech Sound Elements During the First Year of Life: A Review of the Literature," Journal of Speech Disorders, VIII (March, 1943), 115.

Since evaluation of an aphasic's production is very difficult, it might be possible to begin an evaluation of "phonemic regression" by investigating the discrimination ability of aphasics.

Such a study is now underway. A special auditory discrimination task was developed in which 15 consonant phonemes were contrasted. Each phoneme was contrasted with every other phoneme as well as paired with itself in the initial position. These contrasts were developed in word/word, word/nonsense, and nonsense/nonsense syllable lists. These lists were then administered, at a comfortable listening level, to aphasic patients on three successive days. The subject was required to indicate if both members of the pair sounded the same or different to him by pushing the appropriate button.

So far, seventeen aphasic subjects have been tested with these tasks. Other aphasics and a normal population must yet be tested. It is too early to draw definite conclusions. Nevertheless I would like to make some tentative suggestions concerning what we seem to be seeing.

First, two of the seventeen subjects were not able to perform the response task. They seemed unable to remember the task. They gave some evidence of motor perseveration. A highly individualized approach to testing each individual convinced me that they were able to make the discrimination, but were not able to perform the task. These subjects will be investigated further at a later time. For the present, however, we will hold them apart.

Fifteen individuals were able to perform the response task. All of these subjects decreased the total number of errors across the days. This seems to indicate that learning is involved. With the exception of one subject who made 50% errors on the first day, no subject made a large number of total errors in the task. It is difficult to evaluate performance of this group until normals have been tested in the same task. It is interesting to note that the best score made by anyone in this group was a 66 year old woman, classified as global on the Wepman Test.

At this point in our study this much may be said. Most of the aphasics we have evaluated so far have performed creditably on an auditory discrimination task. Those who have not been able to perform the required task have been able to make adequate discriminations. It would begin to appear that one must look beyond lack of discrimination ability for a cause of "phonemic regression."

Linguists and speech pathologists have just begun to explore the possibilities of joint endeavor in studying the many puzzles in aphasia. The language therapist should welcome the guidance that the linguist can provide in describing the regularities and irregularities of language. Careful consideration of the fruits of linguistic research should do much in providing both substance and structure of the therapy process.

EVALUATION OF PROGRESS

Jon Eisenson, William G. Hardy, Martha Taylor

Dr. Eisenson: In evaluating progress, I believe we have to figure out what we mean by progress, and I hope a definition of progress comes from the audience. There are several aspects of this undefined term that I would like us to consider. We tend to assume that a patient has made progress when he is more productive. I think probably we ought to do a little changing, turn the coin over and begin to measure reception of language as a basis for progress. Every aphasic I have ever met has some degree of receptive impairment and until he knows what he is supposed to know; until he is sure of what he is supposed to be understanding we do not know that we have a firm basis for measuring what his production is about. Much of the circumlocution of aphasics--much of what sounds like rather an amazing amount of verbiage on the part of patients who are not quite certain of what the words mean that they are uttering--is a beating-around-the-bush in the hope that perhaps they will know what they are talking about while doing the talking. Maybe if they talk long enough they will understand what it is that they are supposed to respond to. If we re-evaluate the need for productive language on their part, and shift the emphasis to understanding by asking them for a nonverbal act rather than for verbal products to indicate understanding, I believe we might then be in a better position to measure progress.

Another aspect of what I would have in mind by progress is just getting the patient ready for instruction, just getting him ready for therapy. We tend to move in too fast; we are too eager, we are too anxious. I mean that we are anxious in a literal sense. The clinician is too anxious to get started too soon. He wants to feel that *he is doing something*! Perhaps progress can better be measured just in the early stages. That is the kind of progress we make with children when we use pre-primers before we get to the primers, and perhaps we can begin to measure progress in the aphasic by establishing a base for progress in terms of pre-verbal behavior. This is what I have in mind by some of the aspects of progress, but I still want a definition of progress from somebody in the audience.

Dr. Hardy: I have found it necessary on occasion to try to function as a scientist as well as a clinician and one of the troublesome things about this topic, evaluation of progress, is that we simply do not have any aggregate of fact. I know of no single source of information, even at a sampling level, available to any of us to try to answer this question.

There are several more observations one can make. For instance, with reference to definition, it certainly becomes apparent from two or three of the discussants on the immediately previous panel, that, among other things one can say about aphasia, is that as a symptom-complex it offers many aspects for clinical differentiation. I believe we have to learn a lesson from some of the references that Dr. Hoover made, for instance, and Dr. Allan; that nowadays a great deal is being done to remove the complex so that really there is no chronic, long-range, residual problem left. When there is one left, then it offers a whole host of possibilities. I do not see how, for instance, any definition relative to progress can be made except in terms of where a patient was when you began to look at him and where he is later on. And the only way this can be made valid or valuable at all is to develop a descriptive array that gives you a foundation for judgment, and then re-evaluate relative to achievement in the same array. Not only have I never seen two aphasic persons alike, children or adults, I have yet to see any who do not clearly demonstrate confusion on at least the behavioral pre-verbal level: trying to sort out what is going on, what is meant, what is intended for them to do.

In the past several hours, there have been many references to distinctions about severity. I do not believe severity is of much importance in trying to work in the field of aphasia. What is important is to try to designate and differentiate the kinds of problems that different people have. Within the limits of this kind of approach (which is probably most strongly underlined by Hildred Schuell's work and that of the whole Minnesota group), if your classification is reasonably sound to begin with, the question of severity never comes into the picture. It is just a matter of durability on the part of the patient and the clinician. I do not see that we can learn anything by saying this man is in terrible shape and that one just has a little wrong with him. This observation does not help describe the nature of what either may have wrong with him.

We all tend, too, to fall into the trap (it is a big temptation) of trying to over-treat, and that is a point that has been made recurrently. Dr. Eisenson just made it again in a slightly different setting. We do not really know very well, we cannot demonstrate in a very satisfactory fashion, just what connection there may be in serially ordered time, across a fair period of time, between what we do and what happens to the person. The fact that we keep trying to do something does not necessarily have any direct relation to him or to his achievements. We have few facts about the nature, the scope, the spread of the problem. We do not know what happens to the adult who has had all sorts of disturbances after he is released from one treatment center or another. It seems to me that the only people, plausibly, who could produce these kinds of evidence are you most directly concerned with vocational rehabilitation. You have both the means and, I would guess, the interest to try to pursue this kind of question. We do not have any general facts. I do not believe that is an overstatement.

Then there is another dysfunction that enters into this picture: that one oftentimes finds himself having to do business in a milieu, in an environment, which has been framed by particular kinds of administrative policies and principles and which cannot be disturbed. There are so many things that go on for evanescent reasons, nobody ever really knows why. Part of it is habituation, part of it is laziness, part of it is stupidity, and one can add many other parts. I am sure the generalization is true, however, that many of the things we all think of doing (and probably fairly wisely), relative to the kinds of problems we work with, we are just not permitted to do. It just is not in the time scale, is not in the sociology of management within a particular setting. And this is especially true, mind you, in a general voluntary hospital which is designed primarily for the management of acute problems.

I do not have any idea how many strokes, accident-traumatized, and tumor cases come through my own institution in a year. I would guess that it is several thousand. I doubt that we see one-tenth of one per cent of them. I think this is a fairly conservative estimate. I do not know what happens to them. More important, the physicians who treat them probably do not know what happens to them. They just go away, after the acute stage is passed, and somehow or other get absorbed or resorbed in their family circles and muddle along. Eventually, maybe much later on--far too late to be beneficial--somebody interested in general concepts of rehabilitation finds them and tries to do something with them. Actually, these various measures of retraining, or relearning, or reconstitution (whatever you want to call them) ought to be begun within about 72 hours or so after the onset of whatever it was that caused the problem. Because, by that time, any internist who is controlling treatment usually knows perfectly well whether the patient is going to live. If he is going to live, he is going to have problems--and these problems are not going to be resolved by acute hospital treatment. They have to be met, and preferably early, by somebody who is interested in these many manifestations and many ramifications we have all been talking about.

So let me come back to the negative beginning. I have not the remotest idea how to evaluate an aphasic person's progress in all these regards, except in fairly obvious terms of description, relative to status of achievement, in terms of what you thought you saw the man do or not do when you first began to work with him.

Dr Eisenson: Let me respond a bit to something Dr. Hardy suggested and some ideas I have about progress that should not be so discouraging and may be more on the positive side. The conventional way, the established way, for Americans to measure progress is to get numbers and to get quantities. If we think a larger number is better than a smaller number, then if we get a larger number the second time, we have progress. Sometimes you can get the same number, the same quantity and quality of responses, but you get it in less time. Sometimes you can get the same quantity, but with less expenditure of energy or with less concomitant undesirable behavior. If a patient is presented with an inventory, with a standard test of intelligence, with a standard achievement test, or with parts of an achievement test--use any device that you will--and he gets correct answers (let us say he gets 27 points of correct answers in 43 minutes), then you have some kind of a measure. However, you should also measure how much perseveration he had; how much catastrophic reaction he had; how much palm sweating he had; how many times he had to use his handkerchief to wipe his brow; how many times a clinician almost fainted at the thought that the patient was about to faint. All of these indicate what the patient went through. Suppose that two months after an initial test he gets the same score, 27, on a retest. But this time, only once is the clinician afraid the patient is about to pass out. There is now very little sweating. There is very little indication of irritability. You now have other kinds of measures of progress. (Dr. Hardy: Now he does it in 20 minutes, instead of 43). He may even do it in 45. But now, he is at ease and he is taking his time, because he is not anxious any more. A patient may well take more time to do the same amount of work when he is relaxed. From my point of view, he is now making real progress--despite the scores or so-called objective measurements.

Mrs. Taylor: The measurement of progress in the aphasic patient is a problem which has plagued us for a long time. No matter what methods of measurement are available to us, we are all faced with the practical task of selecting criteria for determining progress in the immediate clinical setting. Two alternatives come to mind as possible methods to accomplish this. First, we can routinely retest the patient--using the examinations which were administered initially--and compare his inventory of responses, both quantitatively and qualitatively. This procedure, however, cannot be repeated often, since there is the risk that the patient may invalidate the results by having familiarized himself with the test items. The second possibility for measuring progress is to judge clinically whether or not the patient's verbal behavior has changed. The latter method is undoubtedly the most commonly used.

There is no direct, simple and sound procedure known to date which will definitively measure language change in the aphasic patient. We have been using a series of methods at the Institute, however, which we find extremely useful in assessing the changes occurring in the recovery process. These methods involve the routine retesting of the patient, using three types of tests which were administered at the time of the evaluation: a task-oriented language test; a functional communication profile; and the audiometric test.

The language test is a graded, timed, and objectively scored examination which measures performance in speaking, auditory comprehension, reading, calculation, writing, speaking, and imitation. The time measures, accuracy of responses, and changes in objective scores are used to measure verbal change in each of the language modalities under clinical task-oriented conditions. These results reflect, however, only a part of the patient's total communication performance. If anything, the scores and associated measures probably tell us something of the patient's potential for language recovery. They do not represent the patient's functional use of a linguistic process or vocabulary.

We believe that in certain cases the pure tone audiometric test can give us a measure of change. In cases where initial testing is unreliable, we find that a subsequently reliable set of responses to pure tones usually correlates with improvement in mentation. When an accurate assessment of auditory comprehension is difficult or impossible to make because of the severity of verbal impairment, the reliability of the audiometric test often gives us a measure of mentation.

The Functional Communication Profile[1] is the measure upon which we rely most for evidence of progress in communication. (See page 179.) This rating scale attempts to reflect the language usage over which the patient has control in everyday communication situations, as opposed to the task-oriented

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[1] Martha L. Taylor, The Functional Communication Profile (New York: Department of Physical Medicine and Rehabilitation, New York University Medical Center, 1963).

FUNCTIONAL COMMUNICATION PROFILE[a]

EVAL (Blue)	RE-EVAL (Red)	RE-EVAL (Red)	RE-EVAL (Red)
Date ______	Date ______	Date ______	Date ______
M ___ ___% (.)	M ___ ___% (.)	M ___ ___% (.)	M ___ ___% (.)
S ___ ___% (.)	S ___ ___% (.)	S ___ ___% (.)	S ___ ___% (.)
U ___ ___% (.)	U ___ ___% (.)	U ___ ___% (.)	U ___ ___% (.)
R ___ ___% (.)	R ___ ___% (.)	R ___ ___% (.)	R ___ ___% (.)
O ___ ___% (.)	O ___ ___% (.)	O ___ ___% (.)	O ___ ___% (.)
Overall __ __.__%	Overall __ __.__%	Overall __ __.__%	Overall __ __.__%

	NORMAL	GOOD	FAIR	POOR	0	
MOVEMENT						Ability to imitate oral movement
						Attempt to communicate
						Ability to indicate "yes" and "no"
						Indicating floor to elevator operator
						Use of gestures
SPEAKING						Saying greetings
						Saying own name
						Saying nouns
						Saying verbs
						Saying noun-verb combinations
						Saying phrases (non-automatic)
						Giving directions
						Speaking on the telephone
						Saying short complete sentences (non-automatic)
						Saying long sentences (non-automatic)
UNDERSTANDING						Awareness of gross environmental sounds
						Awareness of emotional voice tone
						Understanding of own name
						Awareness of speech
						Recognition of family names
						Recognition of names of familiar objects
						Understanding action verbs
						Understanding gestured directions
						Understanding verbal directions
						Understanding simple conversation with one person
						Understanding television
						Understanding conversation with more than two people
						Understanding movies
						Understanding complicated verbal directions
						Understanding rapid complex conversation
READING						Reading single words
						Reading rehabilitation program card
						Reading street signs
						Reading newspaper headlines
						Reading letters
						Reading newspaper articles
						Reading magazines
						Reading books
OTHER						Writing name
						Time orientation
						Copying ability
						Writing from dictation
						Handling money
						Using writing in lieu of speech
						Calculation ability

ESTIMATED TOTAL SPEAKING VOCABULARY: 0 1-50 50-100 100-500 500-1000 over 1000

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INSTRUCTIONS FOR USE

A. Initial Evaluation (use Blue pencil)

1. Fully color the area from zero (0) to whatever point on the rating continuum you consider the patient's functional level in each activity.
2. Count the number of filled in blocks - except for those in the 0 column - within each of the 5 dimensions (i.e., Speaking, Reading, etc.) and post each sum score in the left hand column at the top of the page.
3. Refer to Instructions for Scoring on the Conversion Table to calculate percentages, and record corresponding percentage scores in the space provided.

B. Re-Evaluation (use Red pencil)

This rating should occur at regular intervals after the initial evaluation. One month intervals are usually considered minimum re-evaluation periods.

1. Fill in additional blocks along the continuum, in those activities in which the patient has made noticeable functional gain, to the point to which you estimate the patient has progressed.
2. In ink, write the date of the re-evaluation on the highest filled in block in each activity.
3. Again, count the number of filled in blocks (both blue and red) in each dimension, and post each total in the re-evaluation column at the top of the page. Refer to the Conversion Table and record the corresponding percentage scores in the space provided.

Repeat these steps at each re-evaluation.

Please Note: In those rare cases where a patient regresses during therapy, it is advisable to complete a new FCP.

CAUTIONS REGARDING USE

A. Children: In using the rating scale with children who have limited or no development of communication function, "Normal" should be equated with "Average" for the child's particular age level in the general population. Where a child has a communication disorder superimposed upon previously normal language development, "Normal" should then be interpreted as being equal to the child's estimated pre-morbid language level.

Please Note: The Percentage Conversion Table does not apply to children's scores.

B. Clinical Experience: The FCP form has been devised for use by experienced clinicians, having access to hundreds of aphasic patients in any given year. The form may have little validity when used by inexperienced personnel having only intermittent contact with aphasics or access to small case loads.

performance observed in the clinical setting. This tool serves our need for measuring progress during the recovery process more effectively than any other. Recently, computer based research on seven years of experience with the Profile clearly demonstrates its validity as a measure, and its potential for providing prognostic information.

The rationales for the design of such a measure are obvious to anyone actively working with aphasics. An endless number of examples could be given to explain the disparity that exists between language test results and actual language use. We have all shared the experience of administering a test to a patient in which it appears that under task-oriented conditions the patient demonstrates virtually no ability to comprehend spoken language. Yet, the same patient then asked during the examination "Did you bring your glasses?" immediately reaches into his pocket and puts on his glasses. Or the patient who cannot do the simplest calculations, but who still avidly follows stock market activity and transacts the purchase and sale of stocks. Many aphasic patients retain control over gesture and pantomime to the degree that they can communicate a great deal of information by visible signals. Yet these patients would suffer tremendous failure in any standard "aphasia test."

The Profile, then, attempts to rate those dimensions of communication performance which are not usually accounted for in language tests. The basis for scoring is derived from actual use of language skills in a natural context of use, as opposed to the test situation. The Profile was designed to encompass as much information about a patient's actual language function as possible in the simplest, most visible form.

The Profile consists of a list of 50 items (see page 179) considered common language functions of everyday life. Ratings of these behaviors are made on a continuum along an eight point scale, on the basis of informal interaction with the patient in a conversational situation. In this context, the examiner observes and rates what functional use the patient makes of his residual communication skills. "Normal" represents the patient's estimated pre-morbid level of language proficiency. This baseline for "normal" varies of course from individual to individual, depending on many factors; educational, social, and personality factors contributing most significantly. The examiner's ratings take into account the present estimated (1) speed of performance, (2) accuracy of performance, (3) consistency of performance, (4) voluntary control of performance without benefit of external cues, and (5) compensatory function for the behavior (i.e., a patient who cannot say the numbers of the floor to the elevator operator, but who can point to the appropriate floor number where necessary, would be rated as functionally adequate although not "normal"; despite the fact that he must resort to a compensatory method for communication).

An item is rated as "Normal" when it is estimated that the patient can act in the behavior precisely as he did pre-morbidly. This means without any cues, loss of speed, or artificial conditions. The ratings made for each item have weighted scores which are converted to percentages in each of the five modalities: speaking, reading, understanding, movement, and a miscellaneous category which includes writing and calculation. There is also provision made for an Overall Score, which is a reflection of the sum of all of the scores. The Overall Score can be used as a single measure of an individual's communication effectiveness in everyday life.

The ratings are indicated by filling in the blocks, along the eight point continuum for each item, in blue at the time of evaluation. Changes in functional ratings are indicated in red, at monthly intervals, with the date of entry written across the highest red marking. This color coded profile permits the clinician to see the course of change across time in visible form. It also gives the clinician a frame of reference for a "norm" for each individual patient, based on a pre-morbid estimate. An advantage of this type of rating scale is that it makes no reference to diagnostic labels, therefore leaving no margin for ambiguous use or comprehension of terms. The physician or other team member involved in the patient's care can determine at-a-glance (1) where a patient ranks in reference to his pre-morbid communication effectiveness, (2) whether or not a patient is making functional progress in communication, and (3) whether or not a patient can cope with the communication demands of life at home or on the job. Since many language tests are so designed that patients with virtually no language function cannot perform any items, the Profile permits ratings at the lowest levels of performance. For example, the patient who uses meaningless jargon but obviously makes attempts to communicate, could be rated on this scale.

We feel that the Functional Communication Profile has provided a measure of certain dimensions of language performance heretofore not accounted for in the usual language examination procedures. Its effectiveness as a measure of recovery of communication function is becoming more apparent as we gain experience in its use.

DIRECTIONS
IN
RESEARCH

184-186

DIRECTIONS IN RESEARCH ON APHASIA

Fritz Dreifuss, Harold Goodglass,
Martha Taylor, William G. Hardy, Jon Eisenson

Dr. Dreifuss: As a neurologist, I feel that I should recapitulate a few things from the neurological point of view. Until fairly recent years aphasia was more or less the province of neurologists, and whether they got very far in the study of aphasia or not remains still to be determined. Certainly, I think that getting together with psychologists, speech pathologists, psycholinguists, and linguists has expanded the area of research in aphasia tremendously, and to a most valuable degree.

I would, however, make certain warning sounds. First of all, the necessity for observation rather than inference. Not every observation need be attended by inference at the time the observation is made. Secondly, the warning against this structure--this tower of Babel--we are building, where none of us understand the other. People in the field of aphasia go in for neologisms in a big way, probably because they find it so difficult to explain in standard English what they mean. I think it ought to be kept down to a minimum. Thirdly, neurologists from before the time of Broca began drawing diagrams of the cerebral cortex, and every area of cerebral cortex was assigned a certain function; this, of course, we know is not strictly so. In our reaction against this, we should not say there is no such thing as cerebral localization. We should say there are areas of the cortex which are specialized along certain lines, which are necessary for certain actions to take place. We must not replace the maps of the cerebral cortex with maps of what we consider speech to be, such as diagrams of function rather than diagrams of anatomy. Each psycholinguist draws his own diagram of his own concept of what language is, just as each neurologist drew his own cortical map once upon a time. Thinking through a concept is sometimes more valuable than drawing a diagram of it. Fourthly, we ought to expand our concepts to allow of extrapolations in related sciences. For instance, the question of the significance of handedness. It was once thought that cerebral dominance was localized in the left cerebral hemisphere in right-handed people, and in the right cerebral hemisphere in left-handed people. Then it was found (particularly through the injection of Sodium Amytal into the carotid artery and extirpation of epileptic foci) that this was not strictly true, and a complete reaction to this particular scheme occurred; to the extent that we heard it said here the other day that there were odds laid on how often this occurred--how often dominance was in fact in the right hemisphere in left-handed people. I think that if we looked at this very carefully there should not be any great difficulty about this, because left-handedness is due to different causes. There are different types of left-handedness and to say that a certain percentage of sinistrals have left cerebral dominance is not strictly true. There is such a thing as genetic left-handedness, and there is such a thing as acquired or obligatory left handedness, because of damage to the opposite hemisphere. Therefore, one ought to sub-divide one's left-handers into genetic sinistrals and sinistrals who are obligatory sinistrals, because of damage to the left cerebral hemisphere during or after birth. In this way we can make a much firmer estimate than a bet as to how many people are going to be left-handed with right cerebral dominance and how many people are going to be left-handed with left cerebral dominance.

Further, in the study of aphasia, I would emphasize again the very careful study of individual patients, particularly those who have progression of aphasia and resolution of aphasia, in other words, a transient aphasic deficit with the ability afterwards to paint a fine word picture of what happened during this time. Aphasia is found sometimes to progress through various stages and a patient who, at one moment during his migraine attack or during his Todd's paralysis, may demonstrate a syntactical aphasia, may ten minutes later demonstrate a semantic aphasia, and the return from different depths of aphasia, as it were, may occur in reverse order. We intend to do some of this work with Sodium Amytal injections into the carotid artery and actually test these patients as they are going through these various stages, both during the ingravescence and during the return.

Another subject which should be gone into a little further is the question of polyglot aphasia, which really has not been very much discussed. Here there are again many findings, and many inferences drawn from these findings. For instance, some polyglot aphasics obey the rule that the native language--the one first learned--is the one preserved. Sometimes it is preserved to the extent of being grammatically correct, while the secondary acquired language is completely disintegrated. Or, they may behave according to the rule that the secondary language--the one most used in their environment--is the one that is preserved, and the native language or the mother tongue is the one that is lost. These are interesting observations, and I do not think we can make rules as to what is going to happen. This is a very interesting field for further study.

Finally, I would mention cortical stimulation studies. The normal nerve action potential which is generated, and which is conducted, is a function of something in the order of 80 or 90 millivolts. For cortical stimulation, a great big electrode is applied to the cortex and then the cortex is blasted with several volts of electricity which spread every which way across the cortex, through the cortex, down pathways which are not normally used. This is just a travesty of normal function and to make a physiological interpretation from this travesty is incorrect. It gives us some idea, but I do not think that we can, on the basis of crude vocalization evoked in such a crude manner, draw any definitive linguistic conclusions.

Dr. Goodglass: I would like to start by making a plea for more careful study of language processes in individual aphasia patients. This is a neglected area in American aphasia literature, where we see too much reporting on groups of aphasics, without an adequate refinement of tests, based on careful individual case studies. It seems to me that what goes into our aphasia tests is still at a fairly primitive level. We are suffering from a premature application of high-powered statistical techniques to a very inadequate measurement and description of what has happened to the language of these patients.

Since the 1860's, a major dichotomy in patterns of aphasic speech has been recognized by most neurological authorities and represented under many varieties of nomenclature. The non-fluent aphasic, referred to in the classical literature as "motor" or "Broca's," was called a "verbal" aphasic by Head, "predominantly expressive" by Weisenburg and McBride, and "syntactic" by Wepman. The fluent aphasic, classically referred to as "sensory" or "Wernicke's," has been called "syntactic" by Head, "predominantly receptive" by Weisenburg and McBride, and "pragmatic" by Wepman. The differences to which these clinicians responded are: (1) apparent fluency of production; (2) ease of articulation; (3) use of grammatical sequences; and (4) ability to find names for concepts. We can say fairly that none of these features of speech is adequately measured in our current aphasia tests. Let me explain why such measurement is a difficult feat to accomplish and why the superficial approach has not produced useful results.

Consider the problem of articulation. Given that the motor aphasic strikes us as awkward in his articulation, how can we quantify this characteristic in the form of a test score? Currently, we give him sets of words to repeat that require various consonantal sounds in initial, medial, and final positions. We have him do these tasks by repetition preferably, because if he has word-finding difficulty, we cannot depend on his producing the required word; either on showing him a picture to name, or on showing him a written word to read. Our typical, awkwardly-articulating aphasic may not make errors that can be called phonemic errors. His speech is labored, but when you try to put your finger on a specific error, you may go down the entire test list without a scorable error. On giving the same test to a fluent-sounding aphasic, you are very apt to find that when he tries to say the word after you deliberately, he is unable to do so. He may substitute the wrong word, or substitute sounds; he may even grope for the sound, like a motor aphasic. The result is that the fluent aphasic may end up with a poor articulation score, while the motor aphasic comes out with a normal articulation score. The score on a test, then, may fail completely to reflect the most obvious clinical impression. The fault is not with the clinical impression, but with our uncritical application of a familiar test procedure where it is not appropriate.

The difficulty of quantifying aphasic defects is even more vexing in relation to word-finding defects. How is it that a patient can appear to produce language fluently and yet, when he is required to say a particular word out of context, he cannot produce it? When given a particular word to repeat, he may appear not to understand it or he may echo it correctly and not be sure he has said it. At the same time, other aspects of language may be well preserved.

How do we measure word-finding deficiencies? We cannot simply take performance on an object naming test, because if we administer such a test to a group of aphasics, they all have trouble. Simply knowing whether a patient named nine out of ten words presented is not enough to determine whether this person had word-finding difficulty, in the sense I have described. The patient who is severely impaired in all language modalities may be able to name only a few, if any, of the test objects presented. However, a low word-finding score in this context cannot be assumed to measure the same disturbance we observed in the patient who may read, write, and converse at a functional level, but who has a profound inability to produce a given word volitionally. The least we can do, in measuring word-finding difficulty, is to use some sort of ratio of the level of word-finding performance to some other severity level, based on other aspects of language proficiency.

In order to approach the measurement of specific defects, we have to be certain that we are not scoring the end result of a complex performance, in which we lose sight of the process we have set out to measure. Thus, we cannot measure word-finding defects by taking a score on a word-naming test. This most central symptom of aphasia--one which differentiates a specific diagnostic type of aphasia--is one we have not learned to measure. The difficulty of defining and measuring articulatory and word-finding disturbances in aphasia is matched by the elusiveness of other aphasic symptoms. Therefore, we have some very elementary accomplishments to master in the objective description of patients' performance. I think we have to begin by putting more stress on individual case study, to discover just what's happening with a given aphasic, so that we can predict what he is going to do under various conditions. We must find ways of measuring precisely what we wish to measure, before putting the results of our tests into a factor analysis.

An entirely different direction of aphasia research is that which concerns cerebral dominance--a very interesting problem area. The slogan that "half your brain is a spare" may be closer to the truth than we are now willing to admit. It seems fairly certain that, in some way, the brain of a child is more plastic than the brain of an adult. The period during which this change in plasticity takes place seems to have been more or less narrowed down to the years between 9 and 12. There are other phenomena which seem related to the change in plasticity; one of them is the fact that children learn and forget new languages very rapidly during their earlier years, but not so easily in late childhood or adulthood. Another is the fact that when children become very severely deafened before a certain age, their speech has to be maintained by special instruction; whereas if a person is deafened in adulthood, his speech undergoes comparatively minor impairment. These are all changes that seem to have some relationship to the flexibility and plasticity of the brain that is going through some sort of a crystallization during this time in middle childhood. Anything we can do to find out in what way the language processes in the brain of the child are different from the language processes in the brain of the adult, may have some bearing on ways of teaching, and perhaps on ways of using the unused right hemisphere in the adult with left hemisphere damage.

There are various possible leads to this problem. One of them is in the apparent differences in the kind of aphasia we see in left-handed people as compared to the kind of aphasia we see more regularly in the usual run of right-handers with left hemisphere lesions. A number of writers have felt that the left-hander, like the child, is more susceptible to at least a transient aphasia from a lesion on either side of the brain and, like the child, is more apt to recover quickly. In other words, on whichever side the aphasia occurs, it is more likely to be a transient one. It has also been noted that certain symptoms which are specific to various forms of aphasia--for example, the occurrence of jargon and paraphasia--are rare in left-handed aphasics. All this suggests that left-handers are not simply a reversal of right-handers.

We know that left-handedness, in most cases, is not nearly as complete and one-sided as right-handedness. So it appears that what corresponds in the brain to left-handedness--that is, "right-brainedness"--is not in most cases anything like a mirror image of the usual "left-brainedness." There seems to be some capacity for high specialization within the anatomic language area of the left hemisphere of right-handers, that is not present in the right hemisphere of either right-handers or most left-handers. In some way this is a less differentiated brain. It learns its language more like a child's brain learns its language, without fine sub-divisions within its geography to carry out special functions. Now this is all pretty much at a conjectural level, with little threads of evidence to contribute to it, but it is an area that needs research, and one that we are just beginning to find new ways of exploring.

Mrs. Taylor: It is encouraging to see that much of the contemporary research literature in aphasia reflects an interdisciplinary approach to the limitless number of research questions which the disability provokes. With the advent of the computer and modern psycholinguistic theory, a more rigorous set of research tools can be applied to the investigation of many of these questions. The fact remains, however, that the investment in aphasia research is negligible when compared with other disabling conditions.

Certain areas of aphasia research attract my particular attention: (1) the precise linguistic description of verbal symptoms; (2) the effects of specific teaching techniques on language learning; (3) the design and use of measures of residual language function; and (4) criteria for the initiation and termination of treatment.

Researchers in aphasia, unfortunately, continue to be bound by a vague and general symptom classification system which does little sorting out of the linguistic features of verbal impairment. We desperately need precise linguistic description of the verbal symptoms associated with aphasia. Until rigorous, interdisciplinary analysis of the verbal impairments related to brain damage can establish parameters of function leading to more precise classification, researchers will only touch upon the periphery of the questions facing us.

We can probably look forward to a time, not so far in the future, when specific measures of language residuals in aphasia will permit a design of treatment in which the sequential presentation of stimuli under certain controlled learning conditions will assure certain kinds of recovery.

Dr. Hardy: I would like to see some of what we have been talking about today re-related to our general experiences, in terms of formal professional training and current professional needs. No doubt, there is a much more refined relationship to be derived between the speech habits that we tend to describe in phonetic utterance and the envelope of meaning, which may be referred to by a linguist as a phoneme, at a conceptual level. Much of the work that has been done in the last 15 or 20 years in the acoustic analysis of these ingredients of active verbal communication, suggests that when we make a phonetic analysis or a phonemic analysis, in a scientific descriptive endeavor, we are interfering drastically with the reality of the situation. The concept of a phoneme should take into consideration every single aspect of successive utterance and its correlations with meaning. It is not just a thing or a gadget. For instance, consider one kind of dichotomy we were all brought up with: the distinction between vocalic and nonvocalic utterance. There is clear experimental evidence, recently reported by Peter Denes, that the vocalic-nonvocalic distinction has nothing to do with the phonetic detail of utterance; it has no status whatever in the active language-speech exchange in communication. Denes proved this in several ways. First, he filtered out all information in the message-bearing system that could possibly reflect phonetic sound power, so that built into the experiment was the removal of any acoustic information that a vocalic utterance had been made. None of his subjects had the slightest difficulty in making all of the necessary distinctions, inclusions, and exclusions. We all tend to keep deluding ourselves that a particular pattern of analysis, which we may have learned early in formal academic training, really has much relationship in fact. We need to study, in much more detail than has yet been done, what is really going on--not at the phonemic or phonetic level, but at the allophonemic or allophonetic level. We need to enlarge the view of the sense of conveyed meaning, in that we are dealing with a constantly mobile, kaleidoscopic, evanescent, changing set of patterns. Its constants are what we put in; they do not exist otherwise except in the language understanding of the person who is talking or listening. I doubt that we spend nearly enough time with many of our adult aphasics (I am certain not enough with the children--either those developing normally or those in trouble) in trying to make these kinds of analysis. I do not know of a single linguistic study, in depth, of children's early language conceptual development. I have just come back from a meeting on the West coast where, among others, there were thirteen national leaders in the field of linguistics. Not a single one of them could cite such a study, nor was a single one of them interested in making such a study; which was of some concern to me.

One other idea. I believe we sometimes forget that there can well be a difference, in our views of our subjects, between a diagnosis and a description of function and impairments. They are not the same things at all. I do not believe most of us could quibble about a general diagnosis. It is only a broad statement of findings in generalization. It gives one a kind of differentiating classification. I think what we have all been talking about is not diagnosis at all, but detailed description of impairment. Of course, this is the essence of the Rehabilitation Codes we have been working on, because it may be almost impossible to get as many as four or five experts in medicine, or in related behavior fields, to agree precisely about details, although they might all agree on the general diagnostic picture.

There are many kinds of inquiry which must be carried out in the milieu of education. They do not belong in a clinic. For example, within the whole range of children who do not read well (for any one of a dozen assignable reasons, and sometimes for multiple reasons), there are those who obviously read very poorly, and probably twice as many who are not quite that bad but who do not come through adequately. There are many of these less severe problems in learning that should be investigated by people in education, with their own methods. In these programs of research, procedures can be set up and followed through--with data that can be validated and repeated--more readily than is possible with these human brains that are so drastically and dramatically upset. If we could get enough information on the less severe problems, from numbers of persons placed formally in education, I think perhaps we would have another kind of yardstick with which to think about the more drastic problems, whether they be child developmental or adult traumatic. I do not believe that the field of education has begun to approach these tasks.

I would like to add one more point. There is a strong tendency, which heaven knows is humanitarian at base (and therefore I guess good), to assume that we can help almost anyone who is in trouble, particularly if he happens to be in trouble in ways that concern us professionally. But the facts of life are that there are many aphasic adults and many mixed-up children who are going to remain in serious trouble; who are not available for redemption, or rehabilitation, or habilitation. It is a serious lesson to learn, and it probably takes years to learn it; but it is true. What much of the inquiry amounts to, at the research level, is to learn better to make these distinctions fairly early, so that we can concentrate time and energy where fruition may come and not expend time and energy uselessly, in frantic efforts to achieve the impossible. We all need more facts.

Dr. Eisenson: I would like to make several pitches for philosophies of research. I think we can detect the directions of research from the splendid papers presented earlier in the day. I must admit to being a bit concerned that we may be getting so exotic that unless we are able to do elaborate investigations--abstruse in concept and recondite in procedure--we will fail to be impressive. I would like to suggest that there are some simple things we can do that would tell us a tremendous amount about our patients. For example, I am investigating auditory memory span, using just simple digits. I am interested in learning not only how many digits a child can remember or not remember, but the nature of his errors. When he recalls, does he recall in sequence and omit a digit, or does he substitute a digit? Does he leave out the first digit or the last digit? Does he transpose in his recall? I think we may get a lot of useful information about this kind of recall--information that may tell something about this individual's productive language as well. To be sure, this is a very simple experiment. I doubt whether anyone can get a Ph. D. on the basis of this. And yet, I do not think we should lose sight of this important kind of investigation.

I have another kind of investigation in mind that the Russians have been doing, in linguistic generalization. Razran and several other people in this country, Luria and several other people in Russia, are interested in linguistic generalization. The approach is to condition to a word that has possible phonetic as well as semantic dimensions. For example, let us suppose the basic word is *write*. The phonetic dimension is *right*; the semantic dimension is a word like *type* or *spell*. Suppose we take as another word the basic word to which you condition. Let us take *style*, for which the phonetic dimension is *stile*, and the semantic dimension is *fashion*. Razran and Reiss, in this country, have found that if you do this sort of thing with intelligent adults, they will show more conditioning in the semantic dimension than in the phonetic. But they will show some conditioned spill-over--and I use the word *spill-over* almost literally, because the amount of saliva used was the basis for measurement in Razran's investigation. (Razran is regarded as a wet or a fluent conditioner.) In general, Razran found that conditioning with college students showed more generalized response in the semantic dimension than in the phonetic. Working with children under the same experimental setup, Reiss found that the child showed more conditioning in the phonetic than he did in the semantic; but there was some conditioning spill-over in both directions. Now, I think that if we investigate along this line with aphasic individuals we may get some very interesting insights, and we may even get some information as to the state of progress of the individual. We also have found that older adults without known brain damage, who are a little bit tired and past middle age, tend to reverse the direction of their generalizations. When they are conditioned, they tend to generalize more in the phonetic dimension than in the semantic dimension. I think it is plausible to say that any one of us responding to a word probably also responds to several dimensions of the word. With an integrated mechanism, we are able to bear in mind what the requirements are for what we say or need to say. Under conditions of fatigue, or anxiety, or excess of alcohol, we may contaminate one linguistic generalization product by the other. We call these paraphasic errors, or slips of the tongue. It is just barely possible that aphasic individuals respond in both dimensions at the same time. Therefore, their paraphasic errors are the confusion of two dimensions of generalization which normal people keep in mind and control. Let us see if we can commit conscious paraphasia. Suppose the word is style, we have style and fashion, and we may get stashion as the paraphasic error. If we look at stashion in the light of this kind of investigation, we may get a lot more insight into the possible dynamics of this error. The error may be a contamination of a sound; it may just be confusion; we do not really know what it may mean because it may have so many implications. Let us take another word, base; phonetic dimension bass; semantic dimension foundation. Now we may confabulate our own word out of it. Suppose, however, instead of saying that a patient confabulates, we say that the patient is not able to maintain himself--his thinking--in a single dimension. As a consequence of his tendency for linguistic generalization, he still gives us both reactions in a contaminated form. With continued training and continued conditioning experimentation, we may reach the point where we can say that the patient is tending to move in one direction or another. We may induce fatigue; we may introduce drugs, and see if by fatigue or drugs or something else we can switch the direction of the linguistic generalization of a patient. Compared with approaches of some recent investigations, this one is almost ingenuous. Yet, I do not think we should lose sight of this approach and this kind of investigation.

There is one other observation I would like to make on the philosophy of experimentation. This holds for almost anything we can do. I wish it were possible for institutions to say to one another: "We have a notion that we would like to have several other institutions investigate with us concurrently, in this country and in other countries." Then we could learn if the several teams get comparable results or different results. We have to wait, now, for many years before a study is replicated. With concurrent

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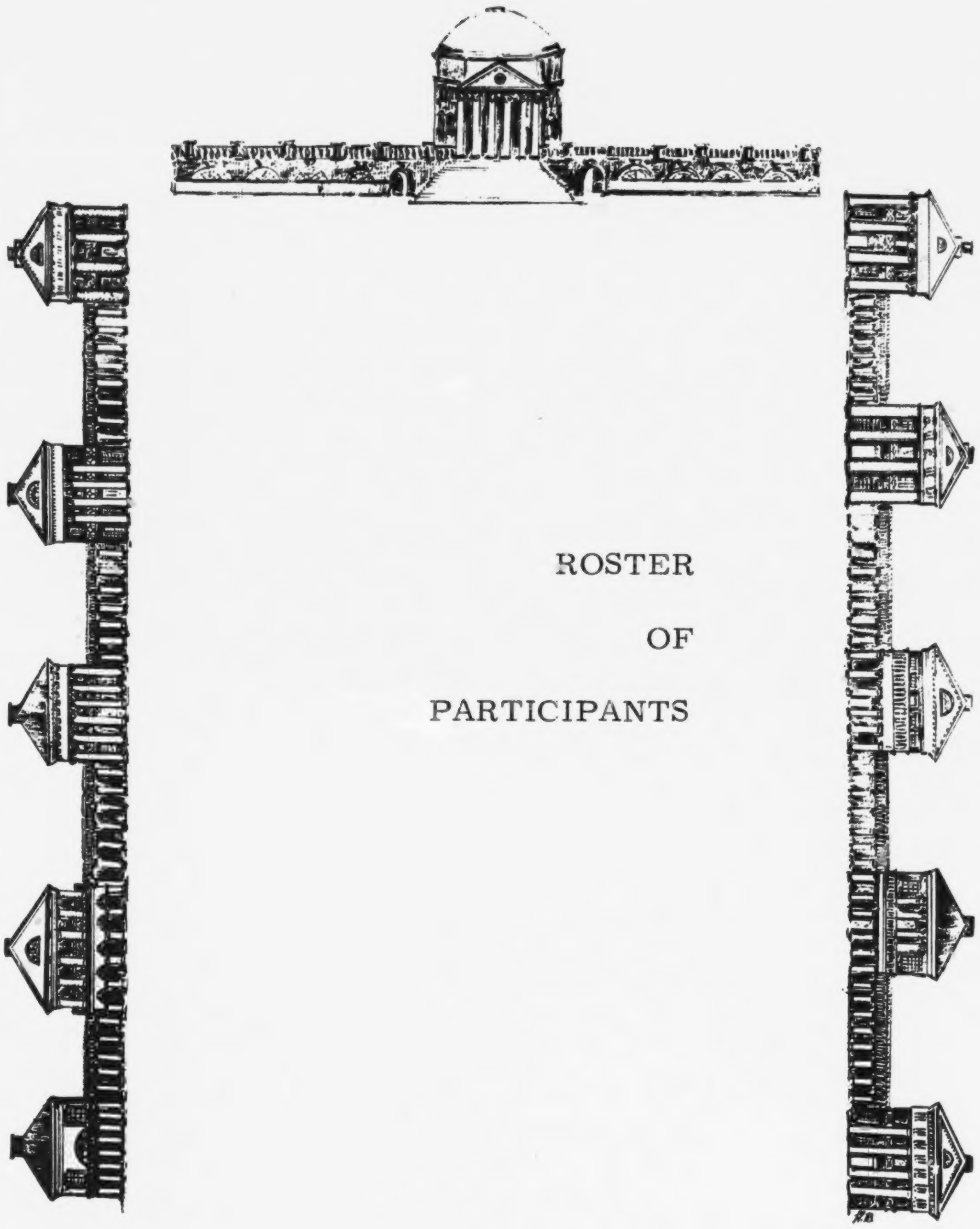
investigations of a given problem--despite our different concepts of the problem--if similar results are found, we may be confident of our answers and the implications derived from the results.



ROSTER OF PARTICIPANTS

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ROSTER OF PARTICIPANTS

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